THE PHYSIOLOGY of DEATH FROM TRAUMATIC FEVER

JOHN D. MALCOLM

Presented by the Author.

THE

PHYSIOLOGY OF DEATH

FROM

TRAUMATIC FEVER

A STUDY IN ABDOMINAL SURGERY

BY

JOHN D. MALCOLM, M.B., C.M.

FELLOW OF THE ROYAL COLLEGE OF SURGEONS OF EDINBURGH, ETC.
SURGEON TO THE SAMARITAN FREE HOSPITAL

LONDON
J. & A. CHURCHILL
11, NEW BURLINGTON STREET
1893

CONTENTS

An Abstract of this Work appeared in the 'Lancet' for February 25th, March 4th, and March 11th, 1893, and in the 'Transactions of the Medical Society of London,' Volume XVI.

THE PHYSIOLOGY OF DEATH FROM TRAUMATIC FEVER

THE CONDITION OF THE VESSELS DURING INFLAMMATORY FEVER

In the clinical study of traumatic fever many facts may be observed which indicate that marked disturbance of the vascular system may be associated with a very slight rise of body temperature, and, conversely, that a decided elevation of the thermometric record may occur conjunctly with no very evident alteration in the pulse rate. Our knowledge of the causes of such combinations of conditions has been greatly advanced by recent investigations into the physiology of heat production and of the increased temperature of the body during the febrile process. The development of heat in living structures depends on tissue changes, and it has been shown that the temperature of the body may be raised at will by the stimulation of a portion of the brain substance to the "medial side of the corpus striatum

near the Nodus Cursorius of Nothnagel."* The conclusion has been deduced that by the stimulation of a particular nerve area in the situation named, "the thermogenic function of the muscles is abnormally increased, and therewith their catabolic or oxidative metabolism; and this without encroaching upon the motor tract, without exciting the motor function, and without any action that can fairly be called vaso-motor coming into play."† With the production of heat a mechanism for its elimination is intimately associated; and there is reason for believing that the heat-eliminating function is also under the control of the thermal nervous system.‡

These conclusions, deduced from the results of experimental inquiry, are so completely in accord with those derived from clinical observation as to justify us in regarding the vascular conditions and the thermal conditions which are found during a traumatic fever as independent effects of one cause.

The Local Vascular Changes in an Inflamed Part.

Before proceeding to analyse the clinical symptoms of a traumatic fever it is necessary to take

* Macalister, "Gulstonian Lecture," 'Brit. Med. Journ.,' 1887, vol. i, p. 670.

† Loc. cit.

‡ Loc. cit.

into consideration some of the local phenomena which occur in the course of an inflammation.

Our knowledge of the local vascular changes caused by an injury is very shortly as follows :— The result of an irritation of the tissues which does not kill them is the production of a "condition bordering on loss of vitality, but quite distinct from it."* As a consequence of this there is induced an abnormal degree of adhesiveness of the blood-corpuscles in the vessels of the affected part. Hence, although these vessels are dilated, the blood moves more slowly through them, and sometimes an actual arrest of its current results. There is at the same time an exudation of leucocytes and serous fluid through the walls of the vessels into the intervascular tissues, which tends still further to obstruct the circulation. The blood-vessels surrounding the irritated area are also dilated, and in these "one sees the full and rapid and more numerous streams of 'determination' or 'active congestion' which extend over a space altogether uncertain."† Other changes may take place involving the destruction of the parts and the substitution of cicatricial tissue, but if the irritation be not too severe, nor too

* Lister, "On the Early Stages of Inflammation," 'Phil. Trans.,' vol. cxlviii, p. 398.

† Paget, 'Lectures on Surgical Pathology,' 3rd edit., p. 228.

continuous, so that it does not too greatly depress the vitality of the tissues, these have "an inherent power of recovery" without undergoing further change.* Clinical observation shows, however, that before the return to the normal condition begins, the local effects of an injury tend to increase for from two to three days. In a superficial wound—for instance, a clean cut just through the skin—the redness and swelling increase for about that time. In such a wound, if the parts be so situated that the divided edges press naturally against each other, the lips of the incision may almost immediately be observed to become adherent. If the wound be small and if the parts remain absolutely quiet, or if complete quiescence be secured by art, there may be little or no evidence of inflammatory action. But if no special care be taken, the movements of the part or accidental disturbances, such as the friction of a towel or of the clothes, produce an evident degree of inflammation, even although union should take place without suppuration. Under these circumstances, towards the end of the second or third day, there is a bright red halo round the injury, and distinct local swelling. The strongest pressure, even against a bone, will not entirely remove the redness from the injured

* Lister, "On the Early Stages of Inflammation," 'Phil. Trans.,' vol. cxlviii, p. 698.

part when the inflammation is at its height. This "shows that while the blood is in parts still free to move there are some minute vessels completely clogged with it."* On the third or fourth day the redness and swelling begin gradually to disappear, and after a few days more there is no evidence of the wound except the scar.

Every inflammation follows a similar course, progressing for a time, generally till about the third day, and then terminating in resolution, formation of cicatricial tissue, or somatic death, or passing into a chronic condition, with, or without, pus formation. It is evident, therefore, that the effects of an irritation on the tissues may continue for about three days before their recovery begins, or before total loss of their vitality takes place. The quieter the parts are kept the less evident are the signs of inflammatory action. On the other hand, every kind of disturbance which may increase the irritation makes the local inflammatory area more extensive. Movements of the parts, by inducing fresh tearing and wounding of the healing tissues, are certainly causes of local spreading of inflammation. The temporary obstruction to the blood-flow must be an important mechanical cause of the increase of the signs of inflammation as distinguished from increase of the irritation itself. When a river is

* Lister, loc. cit.

covered with leaves, if one leaf be caught by a plant or overhanging branch, it is quickly surrounded by others, and a large accumulation may form. In a similar way, so long as there is an obstruction to the circulation, the aggregation of corpuscles and the exudation of leucocytes and serum will remain and may become greater. The blood being impelled against the irritated area, many of its corpuscles are brought in contact with, and consequently within the influence of, the partially devitalised tissues. There is a tendency for these corpuscles also to assume an abnormally adhesive condition, to be arrested in their course, and in turn to affect others. Thus the area of stasis and of exudation enlarges until the tissues begin to recover their vitality, when a free circulation is re-established. Not only does the impulse of the blood derived from the heart's action tend to increase in this way the area of stasis and exudation, but it must also act as a stimulus by, as it were, *hammering* upon the injured part, an evil effect which at once becomes obvious in the throbbing induced if there be a slight obstruction to the flow of blood through an inflamed limb. The local dilatation of the vessels must permit a full stream of blood to impinge on the area of stasis. If, however, a drainage-tube be placed in a wound, we find that the tendency to spreading of the local œdema accompanying

the inflammatory condition is greatly diminished. The escape through the drainage-tube seems to act as a kind of safety valve, and thereby the effects of the unusual force of the blood-stream are to a great extent controlled. Serous matter escapes from the divided surfaces instead of being forced into the surrounding tissues, and enlarging the area in which the blood-flow is obstructed, as in cases where there is no drainage. Consequently the local signs of inflammation are greatly diminished, if not altogether prevented, by drainage. These considerations explain why, during an inflammation, rest of the whole body, and also of the mind, is so much more beneficial than mere restful fixation of the affected parts, or than rest of the body, but with a worried and excited mental condition. If an arm, for instance, be inflamed, and the patient be allowed to go about, even if the whole arm be absolutely fixed, there must be a frequent changing of intra-vascular pressure around the inflamed tissues. When the patient is in a comfortable state of repose the heart-beats tend to be equable and gentle; when he is doing muscular work every contraction of the heart may send the blood violently into the inflamed area, packing the red corpuscles tightly into the obstructed vessels, and forcing the leucocytes and serum into the tissues. Under the latter circumstances an increase of local

signs of inflammation is not to be wondered at. Similarly, variations in the heart's action may be induced by undue nervous excitement, and this seems to be, in part at least, the explanation of the fact that the healing of a wound may be interfered with by mental as well as by bodily restlessness. A full meal and the unnecessary use of stimulants during an inflammatory attack are often observed to have an injurious effect which may be explained in a similar way.

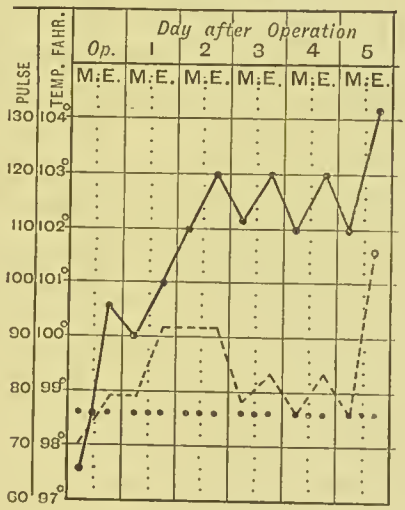
As the injured tissues recover from their lowered vitality the blood-flow is re-established. All exudations are absorbed or are transformed into cicatricial tissue. Everything returns as far as may be to the normal condition.

The General Changes in the Vascular System during an Inflammation.

While these local changes are taking place changes are also observed in the vascular system in other parts of the body. Contraction of the arteries occurs in advancing traumatic fever. After an operation, if union by first intention be obtained, and if the convalescence run a typically healthy course, there is usually some rise of temperature and a corresponding rise of pulse, which reach their greatest height on the second or third evening following the operation. The

rise of temperature and pulse is more continuous and uninterrupted, both in its ascent and in its descent, the more the convalescence is undisturbed by complications. It is usual to have the temperature and pulse rising and falling together, and it has been approximately estimated that, in fever, an increase of the temperature of 1° F. above 98° F. corresponds with an increase in the pulse of ten per minute. But although some such ratio is usual, a great diversity from this correlation of pulse and temperature may be

CHART I.*



found. In some cases the pulse remains slow when fever, if estimated by the temperature, is

* The first two columns of the charts are so arranged that if the pulse and temperature should rise in the proportion of ten beats for 1° F., a single line or two parallel lines would record them. The interrupted line indicates the pulse.

high. For instance, the pulse may range between 80 and 90, and the temperature between 102° and 103° F. In the accompanying chart, No. I, there is exhibited the temperature and pulse during the first five days after the removal of a tumour by abdominal section.

In cases such as this, when the pulse remains slow in spite of high temperature from febrile reaction, the character of the pulse is sometimes much altered. At the wrist it may be full and hard, or small, feeble, collapsing, and easily compressible. When the latter condition exists with a slow cardiac beat, if the large arteries in the neck be examined, the impulse in them is frequently found to be full and bounding. Moreover, when the condition of the heart is investigated it will be observed that the first sound at the apex is loud and distinct, while at the base the second sound is sharply accentuated, or perhaps reduplicated. In such conditions the heart is beating powerfully while the pulse is small and feeble. Hence we must conclude that, although a small weak pulse may of course be due to debility of the heart, yet feebleness of the pulse *at the wrist* is not necessarily an indication of cardiac weakness. This is an important fact both from a clinical point of view and as an aid to the understanding of the pathological and physiological changes which are taking place.

The explanation is simple. The consequence of a greater opposition to the flow of blood through the arteries must be an increase of the work of the heart and of the pressure in the large vessels. It does not follow that the blood-pressure in the smaller vessels is also increased. On the contrary, we know that the resistance to the flow of a fluid through a narrow tube is much greater than it is in a wide tube, and that the resistance increases more rapidly than the diameter of the tube diminishes. Hence the blood-pressure falls as its stream passes from the main vascular trunks outwards, and the smaller the small vessels the greater is the fall of pressure in them. Moreover the small vessels have a far greater proportional amount of muscular tissue in their walls than the large ones have, and consequently a greater contractile power. Therefore in the vascular system of the living body, as the small arteries contract, although the blood-pressure in the larger vessels is raised, the friction in the smaller vessels is so increased that the pressure in them may be greatly diminished. In Lister's paper, "On the Parts of the Nervous System Regulating the Contraction of the Arteries," * it is repeatedly recorded that by stimulation of various parts of the nervous system the vessels were so contracted that the blood was

* 'Phil. Trans.,' vol. cxlviii.

altogether excluded. In that case the pressure of the blood within these vessels was, of course, completely removed. The greater the degree of contraction of the vessels, and the further it extends towards those of large calibre, the more marked must be the diminution of pressure in the arterioles and small arteries. Hence the pressure in the large arteries bears an inverse, though varying, proportion to that in the peripheral arterioles. It follows that the force and fulness of the pulse-beats must be taken only as an index of the strength of the heart *relatively to the obstruction induced by the contraction of the vessels between the artery examined and the heart.* The full, bounding, hard pulse at the wrist occurs when the small arteries only are contracted and the heart's action is strong. When larger vessels, such as the radial, are much constricted the pulse in them becomes small and feeble, and the full bounding pulse is thrown back, as it were, into the larger arteries. The evidence of these changes may be made out clinically in those cases in which the heart continues to beat slowly, although the pulse at the wrist becomes small and feeble. In other cases the force of the pulse-beat diminishes while its rate increases in about the ratio of ten beats per minute for each degree Fahrenheit of rise in the temperature, as shown in the chart, No. II, which

is the record of the pulse and temperature during the first week of convalescence from a simple ovariotomy. In yet another class of cases the

CHART II.

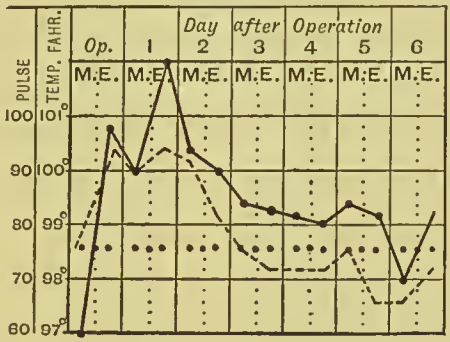
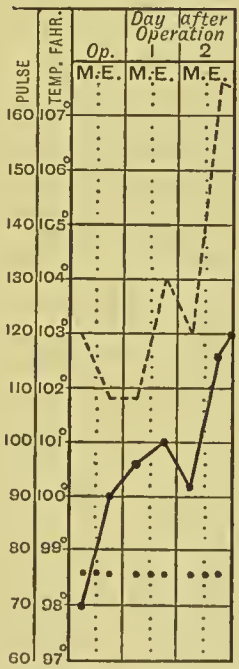


CHART III.*



* The patient from whose history this chart was taken died at midnight on the second day.

increase in the pulse's rate and in its progressing feebleness may be out of all proportion to the temperature. While the latter rises from normal to 103° or 104° F. the pulse may be high immediately after the operation and may go up rapidly to 160 or more, as shown in chart, No. III.

Although cases may be divided into groups in this way, according to the relation which the pulse rate bears to the elevation of temperature, yet many intermediate conditions are observed. The cases in which the heart continues to beat slowly pass by altogether insensible gradations into those in which the heart's action becomes rapid and the pulse weak and running. In this latter condition it is not always easy to make out clinically the evidences of increased peripheral resistance. The impulses in the large vessels may not indicate marked force of the blood current. If these vessels be widely dilated, and the heart-beats follow each other very rapidly, the amount of distension from an individual beat may be small. But the evidences of strong heart action—the distinct loud heart sounds and the forcible impulse in the large vessels—may often be easily made out with a pulse of over 120, if the patient be not too stout. In a thin subject the writer has observed very distinct loud cardiac sounds with a strong thumping of the heart against the

thoracic walls, and a forcible impulse in the large vessels of the neck when the heart was beating 160 to the minute. This was twelve hours before death, which occurred on the third day after an operation. There can be no doubt that in all cases there is the same condition of contraction of the arteries, and it would appear that in severe cases this contraction may affect all the arteries in proportion to the amount of muscular fibre in their walls.

In a typical case of uncomplicated traumatic fever the pulse and temperature rise till the second or third day after the injury, and then gradually return to the normal.

The Relationship of the Local Vascular Changes to those of the Vascular System generally in Inflammation.

We have now seen that, as the result of a wound, there are a stagnation or slowing of the current of blood in the injured part and an exudation of serous fluids and leucocytes around it. This exudation adds to the resistance to the flow of blood, caused by the partial devitalisation of the tissues, and a progressively larger area becomes involved until the damaged parts begin to recover their vitality. At the same time there is a marked and increasing contraction of the arteries throughout

the body. If there be no complication, when the local abnormal condition is recovered from, the evidences of general disturbance of the vascular system also gradually disappear.

We know also that opposition to the blood-flow through any vascular area causes a rise of blood-pressure. This is not brought about merely by the varying amount of blood that is thrown into the rest of the blood vascular system by local alterations of its capacity. The state of the vessels as regards their contraction and dilatation is regulated by the vaso-motor nervous system, whose function it is to apportion the supply of blood to the various organs and tissues. If there be increased resistance in one area, the general blood-pressure is raised by a reflex contraction of vessels elsewhere. Hence, if there be a local increase of resistance to the blood-flow in an inflamed area, there is a physiological mechanism by which a general contraction of the vessels of the body may be accounted for.

But Lister has shown conclusively that around an inflamed centre there is an area of vascular dilatation. The increase of rapidity and of volume in the blood-flow is one of the most obvious changes in the web of the frog's foot near an irritated part. Through this area the blood can pass with so much ease that, if there were no contraction of the vascular system generally, the

effect of the local conditions brought about by an inflammation would rather be to diminish than to increase the general blood-pressure. Thus, the local increase of resistance to the blood-flow due to the stasis in the irritated vessels is counteracted, and therefore it cannot be the cause of the general contraction of the vessels which occurs in traumatic fever.

Nevertheless, though the vascular dilatation which accompanies the local condition thus actually tends to diminish the general blood-pressure, the effect produced on the nerves of the inflamed area—on the nerves of the part where stasis exists—must result in an impression on the central vaso-motor system that, in spite of the surrounding unusually active circulation, there is a local want of fresh blood. The intensity of this impression must increase with the extension of the local action. An attempt may be made on the part of the vaso-motor system to satisfy the necessity for an increased blood-supply to any particular area in two ways—namely, by local vascular relaxation, or by a general contraction of the other vessels throughout the body. Both these conditions are produced by an inflammation. Whether it be a local effect, due to irritation of ganglionic* nervous elements in the walls of the

* Lister, "On the Parts of the Nervous System regulating the Contractions of the Arteries," 'Phil. Trans.,' vol. cxlviii, p. 625.

vessels, or whether it be produced reflexly, the object of the local vascular relaxation may well be to admit of a more full blood-stream to the inflamed part. But, however great the calibre of the minute vessels may become, there still remains the central area of stasis and diminished blood-supply. May we not conclude that the continued want of fresh blood in the area of stasis is the cause of the general contraction of the vessels which occurs ?

It may be objected that stimulation of the nerves of an inflamed area could not produce a sufficient increase of action in the vaso-motor nerves throughout the body to account for the marked contraction of the vessels, and the changes in the pulse which sometimes occur when only a small area of inflammation exists. It may be pointed out, however, that an afferent impression producing reflex contraction of the arteries must pass through nerve-fibres having a relationship to the injured tissues similar to that possessed by the nerves which transmit sensations of pain, if not actually through the same nerves. It is certain that the capability of these latter nerves for transmitting pain is greatly multiplied in an inflamed area. Hence, whether there be special afferent vaso-motor fibres, or whether the afferent impulses be carried along channels which also transmit other nervous impressions, it is certain

that the irritability of the nerves implicated in the inflammatory process, and consequently their power of producing reflex effects, may be greatly increased.

It is, on the other hand, believed by many that the phenomena of traumatic fever are always due to the existence of morbid matter absorbed from a wound and circulating in the blood. Even in aseptic conditions it is said that a "hypothetic substance, 'pyrogen,'" is produced from the damaged tissues, and is the cause of fever. In support of this view it is asserted that many of the component parts of the discharges from a recent wound are pyrogenous.* Without doubt, fresh wound discharges, and other substances, if injected into the blood-stream, do act on the vascular system and on the heat-regulating mechanism. Certain substances also, which are produced in putrifying animal matter and in unhealthy sores, if introduced into the system, even in small quantity, either by injection or through a wound, will kill the patient with absolute certainty. These facts are not to be denied, but it is the object of this thesis to show that the phenomena of fever, and now more particularly that the vascular changes, may be explained without assuming the existence of any such poison,—

* Victor Horsley, 'Heath's Dictionary of Surgery,' vol. ii, pp. 367, 368.

that, in fact, the changes which take place in fever may be attributed to local stimulations acting, through the nerves, on the nerve-centres,—a view which has already been ably advocated from a different standpoint by Dr. Hale White.* It may be argued that the direct effects of an injury —the devitalisation of tissue which Lister has called the "primary lesion in inflammatory congestion,"† the blood-stasis, and the exudations into the affected and neighbouring parts—constitute the pathological condition in an inflammation; and that the general disturbance which accompanies an inflammation—the traumatic fever—is the result of reflex nervous changes,—is caused by *physiological* "reactions," necessarily induced in the healthy body by the local abnormal conditions. This is the view taken, and which will be further developed in these pages.

The conditions under which the individual constituents of healthy discharges may be artificially introduced into the blood-stream are widely different from those which exist in the case of a healthy wound. When fever is increasing as the result of a healthy inflammation in a wound, vital action in the immediate neighbourhood of

* "The Heat Centre Theory from a Clinical Point of View," 'Guy's Hospital Reports,' 1889; "The Theory of Pyrexia," 'American Journal of Med. Science,' November, 1890, and other papers.

† Loc. cit., p. 698.

the injured part diminishes. The tissues become partially devitalised, while the blood and other fluids become stagnant. If the parts be kept absolutely quiet, resolution may take place with very little constitutional disturbance. But if local action be severe, all the appearances which may be observed indicate that the exuded matters remain in the tissues of the inflamed part till resolution begins, while beyond the area in which this exudation occurs there is no evidence that the blood is altered directly by the local changes. If the irritation be sufficiently severe, the central parts may actually die and be separated as a phlegmon. Thus, during the advance of a healthy inflammation, it would seem that there is very little if any evidence that new products are thrown into the circulation from the action taking place in the injured part. Absorption of serous matter necessarily occurs if union by first intention results when a wound is closed without due provision for free drainage having been made. But under these circumstances the more quickly the absorption takes place, the less is the disturbance of pulse and temperature. It is when the discharges *are not absorbed*, when they collect between the lips of the wound, or in the surrounding tissues, and remain there, that febrile reaction runs high. It is then that the evidences of increase of the area of inflamma-

tion and of stagnation of fluids in the tissues are most marked; and with the spread of the local process there is a corresponding increase of the febrile phenomena. When inflammation is undergoing resolution, the case is different. The stagnant and probably altered blood and other fluids and solids are taken back into the circulation. The changes resulting from the breaking down and building up the tissues must go on in a somewhat abnormal way until all effete matter is removed from the part, and the scar is consolidated. During this process there are probably unusual chemical reactions taking place locally, and resulting in abnormal chemical products, some of which must pass into the circulation. At this time, however, the contracted condition of the vessels and all the other indications of fever are subsiding.

The Condition of the Vessels during Shock.

A study of the phenomena to which the term "shock" has been given throws light on the question under consideration. Shock may occur from many causes, and is a usual result of wounds. It may be said to be more a part of the phenomena caused by an injury, whether surgical or otherwise, than a complication thereof. The condition is often extremely well marked in cases

of abdominal surgery, when the operation has been prolonged, and when much interference with the peritoneum has been necessary for the separation of adhesions. The symptoms are well known. In a marked case there is a fall of body temperature, most evident in the extremities and superficial parts, but also affecting the internal organs and tissues even if the surface be carefully covered. The skin is cyanosed, or pallor is extreme. The breathing is slow and irregular, with occasional long sighing inspirations. The expired air is distinctly cold. The mental processes are performed very slowly and indefinitely, or the patient may become unconscious. The sphincter ani may relax. Vomiting commonly occurs, and is not infrequently the first sign of reaction. The pulse is generally small, weak, and rapid. But any of those conditions of heart and pulse which have been described as occurring during an advancing traumatic inflammation may be found, with the exception of the large full pulse. This is the point to which special attention is directed, namely, that the conditions of the vessels which may be found in the various degrees of shock, correspond exactly with those occurring during the progress of an inflammation. Hence we conclude that in shock also there must be a contraction of the arteries.

It is very generally asserted that the condition

of shock is due to a paralysis and dilatation of the vessels, especially of those governed by the splanchnic nerve. On the other hand, Victor Horsley found that in three cases of simple shock, in which he examined the body after death, "the mesenteric vessels were not markedly full at all."* In abdominal surgery, although cases of extreme shock are common, yet marked congestion of the abdominal vessels is rare, and extremely rare as compared with the frequency of severe shock, in which condition it may usually be observed that the intestines and omentum are as pale and bloodless as the rest of the body. If the most conspicuous feature of shock be a loss of tone through the whole arterial system,† it is possible that the capacity of the vascular system might be so suddenly enlarged that a condition resembling to some extent that produced by severe hæmorrhage would result. In fact, it has been experimentally proved that, when the splanchnic nerves are divided, the vessels of the large vascular area supplied by them are so relaxed that other parts of the body become more or less anæmic, and the animal may even die from "being bled into its own belly."‡ If, however, such a loss of tone existed,

* Victor Horsley, 'Heath's Dictionary of Surgery,' vol. ii, p. 434.

† Payne's 'General Pathology,' p. 153.

‡ Landois and Stirling, 4th edit., vol. ii, p. 837.

either generally, or in the splanchnic area only, it would be most unlikely that we should find the condition which may be observed in a few well-marked cases of shock, frequently in the slighter degrees, and during recovery from severe shock, and also in cases of traumatic fever—a condition in which the pulse is almost absent at the wrist, but full and bounding in the carotids, while the heart beats strongly with a distinct first sound and an accentuated second sound. The quick pulse, which is usually one of the most characteristic signs of shock, is not always present. Many if not all of the symptoms of severe shock, including the small, scarcely perceptible pulse, may occasionally be observed with a slow cardiac action. In one case, noted by the writer, immediately after an operation an almost pulseless condition at the wrist was found, while the heart was beating forty-eight times to the minute and with great force.

The evidence to be derived from experimental inquiry is eminently consistent with the view that there is a contraction of the arteries in shock as in fever, for the stimulation of any sensory nerve seems to bring about a reflex constriction of these vessels.*

* Lister, 'Phil. Trans.,' vol. cxlviii, p. 611; Landois and Stirling, 4th edit., pp. 835 and 748.

The Effects of Exposure to Cold in Shock and in Fever.

Exposure to the influence of cold after an injury greatly increases the severity of the symptoms of shock. The effect is apparently proportioned to the intensity of the cold almost as much as to the severity of the injury. Indeed, signs and symptoms exactly simulating those of the condition of shock may be produced by exposure alone. During health, cold in moderation acts as a stimulant to the circulation, causing a more free blood-flow, more active chemical changes throughout the body, and an increase of the excretions and of the elimination of heat. The direct effect of cold on living muscular fibre and on exposed vessels is a contraction of the muscle, and consequently of the vessel. This must cause an unusual opposition to the flow of blood through the cooled surfaces. The influence of the vaso-motor system immediately comes into play. This, as has been remarked, may act in two ways,—by causing dilatation of the vessels leading to the parts where the capillary circulation is obstructed, or by inducing contraction of the arteries elsewhere. By the first of these effects—the dilatation of the vessels leading to the skin—we may explain the action of moderate

cold in health. The temporary redness and sensation of warmth when a cold application is withdrawn would seem to be due to flushing of the capillaries from the still dilated vessels, when the unusual opposition is removed. The dilatation of the smaller vessels throughout the body accounts for the increased tissue change and development of heat which occur. This dilatation of the vessels also affords an explanation of the beneficial effect of cold in relieving fever, for the stimulus to dilatation of the vessels produced by the application of cold to the surface is directly opposed to the stimulation to arterial contraction induced by an inflammation. Thus we might explain the *immediate* improvement of the pulse, which, as Mr. Thornton first pointed out to the writer, may often be noted when an ice cap is applied to a patient with high fever. These changes occur in health, and during a healthy traumatic fever. If, however, the temperature of the surface be lowered too greatly, the obstruction in the superficial vessels becomes so powerful that the compensatory dilatation of the vessels leading to the surface is not sufficient to counteract the opposition to the blood-flow induced by the cold. The second action of the vaso-motor system, therefore, comes into play. The vessels elsewhere contract so as, if possible, to force the blood into the skin. Tissue nutri-

tion and tissue change are diminished. Actual coldness, with subjective sensations of chilliness, is produced, and if there be a weakness anywhere, an inflammation is apt to arise—a partial devitalisation of tissue being brought about either directly by lowering of temperature, or by chilling combined with a temporarily diminished blood-supply. There is in this condition an instinctive desire to take exercise or to apply warmth to the skin, as by sitting close to the fire, either of which proceedings tends to help the circulation. If, however, the cold be sufficiently severe and prolonged the condition of the vascular system which occurs in shock—namely, an intense general contraction of the arteries—may be induced, and if shock already exists, it is aggravated. This would seem to be the explanation of the collapse which sometimes takes place when a case of high fever is treated by the cold bath.

In health, cold and heat, within certain limits, do not alter the body temperature. In a condition of shock, on the other hand, the body temperature is lowered by cold and raised by heat, as is invariably the case in cold-blooded animals. Functional activity is also lessened by cold and stimulated by warmth during shock. The heart of the frog, when removed from the body, goes on beating, but is quickly affected by changes of temperature, its contractions being diminished in

force and rapidity by cold, and hastened by warmth. When similarly exposed the young mammalian heart also goes on beating for a considerable time, and, being necessarily in a condition of shock, behaves in precisely the same manner as the batrachian heart when warmed and cooled. If the main vessels of a kitten under chloroform be divided close to the heart, and the latter be freely exposed, its action will gradually become less frequent, and finally will cease; but a steady beat may be again induced by closing the warm parietes over the organ. From this the conclusion is certain that, in warm-blooded animals, the protection afforded to the heart by its position is of great importance in preserving it from the effects of exposure to shock. All the internal parts are protected by similar salutary conditions. Hence, when contraction of the arteries in shock is intense, we may conclude that those parts which are best protected from the cold will maintain the most free circulation. In fatal cases the blood-flow will continue longest in the internal parts, and hence, especially if death occurs slowly, there may be found on post-mortem examination an enormous distension of the abdominal vessels. On the other hand, when death from shock is instantaneous, the whole vascular system, including the heart, being simultaneously affected, the dilatation of the

abdominal vessels will not be found, as was the case in the three examples of simple shock noted by Horsley. Distension of these vessels is therefore not an essential condition, far less the initial cause of shock. If it were so, it must have been frequently observed during life, and described by surgeons. When this condition is found on post-mortem examination, it is not to be regarded as the cause of death, but as a consequence of the mode of dissolution.

It is to be particularly noted that the condition of shock may, and usually does, occur so suddenly that it cannot be supposed that the development of any poisonous substance can take place, and act as a cause of the phenomena observed. Therefore the contraction of the arteries which occurs in shock is certainly due to nervous influences. It follows that in traumatic fever also the contraction of the arteries may be entirely a reflex nervous and physiological effect; and it is much more rational to attribute the pulse phenomena, which are found in traumatic fever, to such a reflex nervous action, produced by a mechanism which we know to exist, than to the presence of a hypothetical poison acting in a manner not known.

It seems, therefore, that in traumatic fever the contraction of the arteries throughout the body may fairly be attributed to a physiological reflex

action resulting from the exclusion of fresh blood from, or diminished supply of blood to, the inflamed area. In this view of the physiological effects of inflammatory action we have a full and adequate explanation of the vascular changes induced. So far as these are concerned, the action assumes a rational unity. There is an obstruction to the blood-flow in the irritated centre, and a local deficiency of fresh blood. There is a reflex contraction of the vessels in the other parts of the body. Between the local and the general increased resistances there is an area in which the blood-vessels are dilated, and which surrounds the partially devitalised centre. Through these dilated vessels lies the direction of least resistance to the blood-current. Through them, therefore, the blood-stream flows in greater volume, and the excess of rapidly moving blood thus brought into the neighbourhood of an injury has been called the "active congestion," or "determination of blood" to the part.

The Self-restraining Effect of a Universal Contraction of the Vessels.

The vaso-motor system must also exercise a restraining action on the extent of the vascular contraction produced. The constriction of the arteries consequent upon an inflammation neces-

sarily diminishes the supply of blood to the tissues generally. If the supply of blood becomes too scanty, messages will be conveyed to some central nervous area from all parts of the body, indicating with increasing force that the supply of nutriment to the tissues is being unduly diminished. There must thus be created an almost universal reflex stimulus to dilatation of the vessels, which will act in opposition to the abnormal stimulus to contraction emanating from the inflamed area. Thus, at any moment, the state of the arteries during a traumatic fever depends on the force of the abnormal stimulus to contraction caused by the local devitalisation of tissue ; but this force is more or less limited by the counteracting influence arising in the healthy parts, and tending to induce the normal degree of vascular relaxation. The patient's condition is, so to speak, hung in a balance, and it is easy to understand how, in severe cases, an apparently small matter may turn the scale one way or the other.

The Effect of Constipation and Obstruction of the Bowels on the Condition of the Vessels in Traumatic Fever.

It is well known that if constipation be permitted to continue after any operation, the signs of fever are likely to become more evident, and that the

febrile exacerbation may almost certainly be alleviated, temporarily at least, if not permanently, by the action of a purgative. In abdominal surgery it sometimes happens that serious general disturbance and even death is induced by retention of the contents of the bowel after an operation. The symptoms brought about by this complication seem to have been very generally mistaken for those of peritonitis. The diagnostic signs which distinguish inflammation of the peritoneum from a simple retention of flatus, due to paralysis or obstruction of the bowels after an abdominal section, do not appear to have been differentiated until the writer published a paper* on this subject in the autumn of 1887. About a month after this paper was read before the Royal Medical and Chirurgical Society of London, Professor Olshausen made a communication to the Obstetrical and Gynæcological Society of Berlin,† in which he advocated similar views. He, also, described how paralysis of the intestine may give rise to symptoms of obstruction. The condition was called a "pseudo-ileus," and it was claimed that this was a "hitherto undescribed cause of death from laparotomy." Six

* "On the Condition and Management of the Intestines after Abdominal Section," 'Med.-Chir. Transactions,' vol. lxxi.

† 'Centralblatt für Gynäcologie,' January, 1888; and 'Brit. Med. Journ.,' May 19th, 1888, p. 1073.

months later, in the 'Revue de Chirurgie,'* Dr. F. Verchère also published a description of death from paralysis of the intestine following abdominal operations. He, too, stated that this cause of death had "always been confounded with peritonitis." The conclusions which were come to by Olshausen and Verchère differed widely, however, and in most important respects, from those which have been arrived at by the author of this paper. The subject is, therefore, worthy of further consideration; and this is a convenient opportunity for discussing the question, as the conditions found in cases of pseudo-ileus throw light on the causes, and also on the effects of contraction of the arteries in inflammation.

When an operation is performed, which necessitates the opening of the peritoneal cavity and the manipulation of its contents, there is obviously a considerable likelihood that the various coils of intestine may be disarranged, and may be left in such positions that the passage downwards of their contents will be hindered. The peristaltic action of the intestines usually unfolds any awkward turns which may have been thus produced in them. But in many cases, particularly when adhesions have been separated, it is impossible to avoid leaving patches or tags of raw tissue. If any such

* July, 1888.

have been left in contact with coils of intestine, adhesions between the cut surface and the healthy peritoneum may take place with very great rapidity; and if there have been any mal-arrangement of the parts, the bowels may quickly become so absolutely fixed in their new positions that only considerable force will release them. Thus there is always a risk that a mechanical obstruction of the bowels may arise unless a healthy peritoneal surface is everywhere made absolutely continuous. In one case operated on by the writer in the Samaritan Hospital, death was due to adhesion of bowel to a very small surface of a pedicle of the broad ligament. A complete intestinal obstruction was produced without any sign of peritonitis being found after death.* Similar conditions have been observed in other cases.

In the exposure and manipulation of the intestine there is also abundant cause for its temporary paralysis. The muscular wall of the alimentary canal receives its nervous supply in great part from branches of the large abdominal sympathetic nervous centres. These branches terminate in the wall of the intestine in Auerbach's and Meissner's plexuses, which are very rich in ganglion-cells; and the former of them contributes the nervous supply to the fibres

* Malcolm, "On some Complicated Cases of Abdominal Section," 'Lancet,' 1891, vol. ii, p. 120.

of the muscular coat of the gut. The plexus of Auerbach, though under the control of the central nervous system, is also capable of being influenced by any kind of stimulation brought to bear directly on the walls of the intestine.* Variations in temperature, mechanical and chemical stimuli, and alterations of the quality of the blood flowing through the mesenteric vessels, all produce an effect on peristalsis. Every stimulus causes, first, an increase of muscular activity, but when strong or prolonged it also induces paralysis from exhaustion of the nervous energy, which can only be recovered from after a period of rest. It is obvious that during an abdominal section many stimulating influences are brought to bear on the peritoneum, so that some degree of paralysis of the gut is to be expected after these operations. In nearly all cases there are some signs of the existence of this paralysis, while in a few the evidence is strong and unmistakable.

During an operation on the abdomen, peristalsis is rarely seen except for a very short time after a coil of intestine has been exposed. After an abdominal section there is usually, for a time, sometimes as long as two days or even more, no evidence of the development of gases in the intestine or of their propulsion down the gut.

* Landois and Stirling, 4th edit., p. 281.

There is no abdominal distension, and little or no escape of flatus. When gases do form they are often absolutely retained by the sphincter ani, unless a tube be passed through the anus to facilitate their exit, while frequently an evacuation of fæces does not occur for a week, and sometimes for much longer after the operation, unless assisted by an enema or by laxative medicine. These conditions indicate that the activity and power of the peristalsis are diminished for a time. The risk of the occurrence of obstruction of the intestine from misplacements and adhesions is greatly increased by this inactive and weakened state of its peristalsis. An early physiological effect of an obstruction is the production of increased muscular action in the bowel immediately above the affected part. If, however, the muscular power of the intestine be so weakened that it cannot overcome the obstruction, a local dilatation of the gut results, and the bowel is thus placed in a most unfavorable condition for exerting its power. It is, therefore, very apt to continue in a state of inertia or of ineffective action, and the chances of the obstruction being overcome are lessened.

Thus after any laparotomy a pseudo-ileus may result, either from feebleness of peristalsis, or from a mechanical obstruction, or from a combination of these conditions. Olshausen attributes

the paralysis of the intestine, in the cases which he describes under the name of pseudo-ileus, to "the disturbance of the circulation in the walls of the gut which occurs after a prolonged eventration," and to consequent "venous hyperæmia and extravasation in the wall of the gut." These conditions may of course occur, and evidences of vascular changes in the peritoneum are usually found at the autopsy when death is caused by a pseudo-ileus following an abdominal section. Indeed, the changes found usually indicate a well-marked inflammation.

Cases, however, have repeatedly been observed by the writer, showing every clinical evidence of paralysis of the intestine and pseudo-ileus, with signs of peritonitis and congestion of the peritoneum found after death, but in which there was no evidence whatever of congestion or of any structural change in the wall of the gut when the abdomen was reopened during life. The peritoneum appeared perfectly healthy, although, at the time of reopening, the symptoms of pseudo-ileus were so well marked that it was for the discovery and, if possible, the relief of obstruction that the second operation was undertaken. In some cases of this kind an obstruction has been found. In others nothing has been discovered which could be described as an obstruction, either at the second operation or at the post-mortem ex-

amination. In these cases the symptoms of pseudo-ileus were well established before there was any sign of mischief in the peritoneum. Hence neither peritonitis, nor hyperæmia, nor extravasation into the peritoneum could have been the cause of the unfavorable symptoms first produced.

In similar cases when no second operation has been performed, signs of peritonitis are almost invariably found after death. But in some cases which clinically were not to be distinguished from those just mentioned, no evidence of peritonitis or of extravasations into the wall of the gut has been discovered post mortem. There has either been a simple obstruction, or nothing abnormal was observed except the distension of the bowels.

Again, the symptoms of an intestinal obstruction may be well marked and advancing, when suddenly, without apparent cause, or after a purgative enema has been administered, or as the result of a dose of laxative medicine, flatus begins to pass from the anus, perhaps an evacuation of fæces occurs, and all adverse symptoms at once disappear.

The following case illustrates this sudden relief. After a simple ovariotomy everything went on well till midnight on the second day (about sixty hours) after the operation. Flatus had then passed from the anus repeatedly through

the rectal tube, the abdomen was flat, soft, and natural, the menstrual period had come on freely, the temperature had been between 99·4° and 100·4° F. in the vagina all day, and the pulse was steady at 98. At midnight on the second day the tube was, as usual, passed into the rectum previously to the administration of a nutrient enema, and on this occasion only a very little flatus escaped. The patient complained of some pain in the abdomen, which gradually began to distend. Flatus ceased to come down into the rectum, so that none escaped when the rectal tube was inserted. The temperature crept up slowly till it reached 101·2° F. at 7 o'clock on the evening of the third day. The pulse between 7 a.m. and 7 p.m. of this day rose from 96 to 130, and became very small. At 10 p.m. a large gruel enema was administered, and during its injection, after about three pints had been introduced, a loud gurgling sound was suddenly heard, apparently coming from the neighbourhood of the splenic flexure. The writer at once placed the palm of his hand on the left hypochondriac region, and felt distinctly the irregular tremulous vibrations due to an escape of fluid or of gases, which had been confined in some portion of the subjacent bowel. About half a pint of the enema was allowed to return, through the rectal tube, half an hour after its administration, and some

small lumps of fæces came away with it. At 11 o'clock p.m. the temperature was 102·2° F., the pulse was 130. During the night the temperature and pulse steadily descended to 100° F. and 96 respectively at 9.30 a.m. next day. The patient vomited twice in the night, and the nurse stated that on the second occasion some small lumps of fæces were brought up, but the vomit was not preserved for inspection. After the vomiting, flatus passed freely through the rectal tube inserted at frequent intervals. The abdominal distension quickly disappeared, and the patient's progress to recovery was continuous and in every way satisfactory. The great bulk of the gruel enema was retained and absorbed. When the bowels were moved by a clyster on the sixth day after the operation, the stool was very copious, and for several days following there were very large evacuations. It seems to be a fair conclusion that the condition which was threatening to kill this patient was the inability of the peristalsis to propel downwards the contents of the bowel, a mass of fæces having probably got jammed at some twisted, bent, or narrowed portion of the gut, where it caused an absolute obstruction until dislodged by the enema. Cases in every respect analogous to this are not uncommon. Were the conditions observed in such cases due to a gross lesion of the peritoneum like

"venous hyperæmia and extravasation into the wall of the gut," there could scarcely be such a complete and rapid cure as may not infrequently be observed. It is not to be denied that venous hyperæmia and extravasation into the wall of the gut may occur as the result of a surgical, or any other, injury to the abdomen. But when these changes exist to an extent sufficient to paralyse the intestine and produce symptoms of pseudo-ileus, recovery can rarely take place. There are then two conditions present,—the "hyperæmia and extravasation into the wall of the gut" and the paralytic distension of the intestine,—each of which tends to aggravate the other. When a condition of hyperæmia and extravasation into the wall of the gut occurs as a direct consequence of an operation, the affected part is really in a state of inflammation—which may be only in its earliest stage, but is still inflammation. It would appear, however, to have been Olshausen's object, as it certainly was the writer's, to differentiate a class of cases which die after an abdominal section, notwithstanding that the wounds heal well, for the first few days at least, and that there are no undue inflammation of the divided tissues, and no general peritonitis as a direct consequence of the operation. Such cases do occur, and no marked structural change is necessary to produce symptoms of pseudo-ileus. The

inflammation of the peritoneum, the signs of which are so frequently found after death in these cases, is not developed until the symptoms of obstruction are unmistakably present,—indeed, peritonitis may not occur at all—no sign of it may be discoverable after death.

Whatever be the cause of the retention of the contents of the bowel, there can be no doubt that when this becomes absolute during the first few days after an abdominal section, the condition of the patient is a most dangerous one. Under such circumstances, death from pseudo-ileus or from obstruction will certainly result unless relief be obtained spontaneously or by treatment. In fatal cases death is preceded by very definite clinical signs; and the state of the pulse, vessels, and heart in such cases is of great interest in connection with their condition in inflammation and in shock.

In considering these conditions, as in the case of an injury, a sharp distinction may be made, and in the following pages will be made, between the primary abnormal conditions and their effects. Excessive feebleness of peristalsis and mechanical occlusion of the lumen of the bowel are pathological conditions; it will be shown that the results of these conditions may be traced to *physiological* reactions.

In a typical example of death from a simple

paralysis or obstruction of the bowels after a laparotomy, one of the most important points to be observed is that the evidences of changes taking place in the wounded tissues, and the evidences of the existence of traumatic fever, are exactly as if the case were uncomplicated, until an amount of fluid and of gases sufficient to produce symptoms of obstruction has collected in the intestine. If the patient be kept quiet and be not fed by the mouth, distension of the bowel may not commence till forty-eight hours or more after the operation. During a normal convalescence flatus is commonly passed *per anum* before this; but about this time, or soon after, gases are developed in the intestine and expelled from the rectum in considerable quantity, sometimes in very large amount, whenever a tube is passed through the anus at intervals of three or four hours. If this escape does not occur, the cause may be simply that, as sometimes happens, an unusually small quantity of flatus is being formed. But if retention be due to inefficient peristalsis or to an obstruction of the bowels, abdominal distension progressively increasing will occur as the flatus accumulates. This distension is usually unaccompanied by pain, although at its very commencement there may be colicky pains indicating that the bowel is making an effort to overcome some obstruction. As a rule such

pains soon cease, for the already weakened bowel quickly becomes completely paralysed by the distension.

When the enlargement of the abdomen begins on the second or third day after an operation which is otherwise uncomplicated, its first appearance is coincident, or nearly so, with the highest temperature and pulse naturally arising from the traumatic fever. The pulse and temperature, after rising as usual, frequently show a decided tendency to fall again, so that marked and increasing symptoms of obstruction may be associated with the signs of declining fever.

If relief of the obstruction be not obtained there is always a return of the rise of temperature and pulse. This may occur at a very variable period, from a few hours to forty or more before death. The pulse and temperature may rise together, but it is in these cases that the most marked divergence between the extent of the thermal and of the vascular changes are sometimes observed. Whether the rate of the cardiac action rises or falls or remains steady, the pulse at the wrist invariably becomes small, feeble, and compressible as the abdominal distension becomes greater. In a case of this kind, the writer noted that it was quite impossible to count the pulse at the wrist when the heart was beating strongly 108 to the minute.

When the intestine begins to expand, the

vessels in its walls must be lengthened, narrowed, and subjected to pressure. Consequently there is an increase of resistance to the blood-flow in the vessels of the part first affected. If the contents of the bowel do not pass down, the distension of the gut and the increase of resistance to the blood-flow in its vessels become greater, and spread to other parts, until a large portion of the intestine may be tensely expanded. Later, when the abdominal walls are also stretched, pressure is made on the vessels of the parietal peritoneum, and also on the aorta and inferior vena cava. This condition does not, as might at first sight be suggested, drive the blood into other vascular areas. During digestion there is an unusual flow of blood to the stomach and intestines, but it does not follow that there is a diminished supply of blood to other parts. On the contrary, during digestion it may usually be observed that the pulse at the wrist becomes more full and soft. There seems to be a diminished blood tension throughout the body. On the other hand, constriction of the mesenteric vessels from intestinal distension raises the blood-pressure generally. This is brought about not only by the reduction in the capacity of a large vascular area, but also by the reflex mechanism which, as already pointed out, causes a contraction of the arteries in other parts as a consequence of a local opposition to the blood-flow. The general

contraction of the vessels is necessary to equalise the pressure, and prevent the circulation through the intestinal walls from being stopped altogether. Hence, if abdominal distension should occur when the blood-pressure from any cause is already high, it is obvious that the effect on the pulse must be very great. Thus there is a physiological explanation of the small pulse in cases of abdominal distension during a traumatic fever. It may be remarked, by the way, that this interpretation of the conditions found in a case of pseudo-ileus fully accounts for the "thready" pulse of peritonitis, in which disease inflammation and tympanites are usually combined. That the small pulse in cases of pseudo-ileus following an abdominal section is not necessarily due to cardiac debility is shown by a comparison of the pulse with the condition of the heart and large vessels. On examination it will be found that exactly the same relationship obtains between the force of the pulse and the force of the heart-beats as occurs in cases of traumatic fever and in shock, although, as in these diseases, this is not clinically obvious in every instance.

It seems, therefore, that in all these conditions, in advancing inflammation, in shock, and in abdominal distension following a laparotomy, contraction of the arteries is brought about by a reflex physiological mechanism; that this contraction of

the arteries, while raising the blood-pressure in the large vessels, accounts also for the smallness and feebleness of the pulse in such vessels as the radial; and, consequently, that this condition of pulse is due, not to cardiac weakness, but to the relation which the cardiac strength bears to the obstruction produced by the contraction of the medium-sized and small arteries.

The Effects of Contraction of the Arteries on the Rate of the Cardiac Rhythm.

Contraction of the arteries must immediately increase the resistance to the flow of blood through the large vessels. The effect of this on the pulse rate is variously stated by different authors. Foster says that "the relation of heart-beat to pressure may be put almost in the form of a law that 'the rate of beat is in inverse ratio to the arterial pressure;' a rise of pressure being accompanied by a diminution and fall of pressure with an increase in the pulse rate."* On the other hand, in Landois and Stirling's Physiology it is said that when the blood-pressure is diminished, the heart executes extremely small and slow contractions, and conversely that a rise of blood-pressure increases the pulse rate.† These two passages are certainly contradictory of each other.

* 'Foster's Physiology,' 3rd edit., p. 178.

† 4th edit., p. 837.

Professor Roy and Mr. Adami, of Cambridge, have brought forward some interesting experimental evidence on the effects of increased blood-pressure on the heart. They have shown that the pressure within the heart rises with every increase of resistance to the flow of blood through the aorta, but that this intra-cardiac pressure has a limit, which varies in different animals and also at different times in the same animal, according as the heart is strong and vigorous or weak and exhausted from starvation or fatigue.* It has also been shown that in case of injury of the valves of the heart the cardiac muscle does more work, and the mechanical deficiency is thus made good, for a time at least, so that the pressure in the vessels does not fall.† So long as the limit of intra-cardiac pressure is not reached, "the heart goes on contracting and sending out all the blood which reaches it (excepting of course the residual blood)."‡ If the limit be exceeded, sudden cardiac failure results, and unless the resistance to the flow of blood through the aorta be speedily removed, the animal dies.

It is clear that the heart does not in ordinary conditions work at its full power. We know also that in many diseases in which the work of the

* "Remarks on Failure of the Heart from Overstrain," Roy and Adami, 'Brit. Med. Journ.,' 1888, vol. ii, p. 1321.

† Landois and Stirling, 4th edit., p. 69.

‡ Roy and Adami, loc. cit., p. 1324.

heart is increased, as in obstructive diseases of the orifices of the chambers of the heart, in valvular incompetence, and in kidney disease with increased peripheral resistance, when the abnormal conditions come on slowly, the increased work causes cardiac hypertrophy. The reserve energy of the heart comes first into play, and then, in accordance with all experience, the muscle which is regularly subjected to hard, but not excessive work becomes larger and stronger. Under such conditions the heart's action is not necessarily or even usually hastened. On the contrary, so long as compensation is sufficient, the pulse is very generally slow,—a rise of pressure is accompanied by a diminution in the pulse rate in accordance with Marey's law. If, however, the work of the heart be too greatly or too suddenly increased, a quick, feeble pulse results, or instantaneous death may occur.

It is a clinical fact that a weak heart is very apt to be a quickly beating heart. But weakness is a term entirely relative to the work to be done. For instance, we find clinically that a heart which has not previously shown signs of debility is apt to beat very quickly when hampered by pressure, as in pericardiac effusion, or pleurisy with effusion, or when subjected to a sudden and severe strain, as when one who has been long confined to the horizontal position is injudiciously al-

lowed to sit up or attempt to stand. In active exercise also, in which there is a strictly physiological but it may be an extreme condition, the rapidity of the pulse must be attributed to the extra work performed by the heart. So the quickening of the pulse in fever, and in shock, may be attributed to the increase of work thrown upon the cardiac muscle by the contraction of the arteries.

Cases in which the Pulse Rate does not increase in the usual Proportion to the Temperature after an Operation.

Light may be thrown on the effect of increased intra-cardiac pressure on the pulse rate by a study of certain cases in which there is evidence of constriction of the arteries and consequent increase of blood-pressure in the large vessels, but in which no corresponding acceleration of pulse rate takes place. In two cases of pseudo-ileus after abdominal section, the writer detected a very marked increase of pulse fulness and hardness when symptoms of obstruction first commenced, but without any increase of rapidity of the pulse. In the first of these cases there quickly followed a great increase in the pace of the cardiac action, while the pulse at the wrist lost its force, and became more and more feeble as the abdomen distended. The patient died with typical clinical

signs of pseudo-ileus, the diagnosis being fully confirmed after death. In the second case, in which a slow, full, deliberate character of pulse was noticed in connection with retention of flatus and abdominal distension, a purgative was administered during the continuance of the slow heart action. As a consequence flatus was expelled freely from the anus, and the abdominal distension subsided. No quickening or enfeeblement of pulse followed, but its unusual force and fulness at once disappeared.

Such a full, tense, but not quickened pulse frequently occurs in the early stages of inflammatory diseases. There are other cases in which, as already pointed out, there is an increase of cardiac work, and, judging by the temperature and general condition, a rapid pulse would be expected, but is not found. Such a case as the following illustrates well the condition to which attention is desired.

A woman of whose case careful notes were taken had a particularly powerful and healthy heart, as judged by the clinical examination. A tumour was removed from the abdominal cavity by a very severe operation, during which much separation of adhesions to the intestinal and other parts of the peritoneum was necessary. The operation was performed on a Wednesday, and the temperature gradually rose to 102·8° F.

at 11 o'clock p.m. on the following Friday. The temperature after this was steady at 103° F. for thirteen hours. It then fell a little, but on the Saturday afternoon it reached 103° F. again, and remained so for ten hours, and on the Sunday evening it once more rose to 103° F., and maintained this height for five hours. The lowest temperature in the intervening days was 102° F., (the temperature being taken every three hours, and sometimes oftener). The highest pulse rate counted up to the morning of the fifth day—the Monday—after the operation was 92, and the more usual rate was 76 to 84, as shown in Chart No. I, which was compiled from this case. On listening over the heart the cardiac sounds could be heard exceedingly loud and distinct, and the second sound was markedly accentuated at the base. Later on, this patient developed signs of obstruction of the intestines, and the heart beat quickly—at one time as quickly as 130 to the minute. This, however, was after the cardiac muscle had been subjected to a severe strain and to a prolonged period of hard work. The relationship of the temperature to the pulse during the first five days after the operation shows that in this case there must have been some difference from the usual relations of the vascular to the thermal system. The patient ultimately recovered, so that the condition of the heart itself could not

be determined by anatomical examination. The explanation of the phenomena observed may, however, be found in the clinical evidence, detected before the operation, that the heart was an unusually powerful and efficient organ—a fact which became even more apparent on auscultation after the operation.

In another case, in which there was certain clinical and post-mortem evidence that obstruction, or rather a pseudo-ileus of the intestine was the cause of the fatality, the pulse was beating 96 to the minute six hours before death. One hour before death the pulse was felt at the wrist with difficulty, and could not be counted there, but the heart was beating 108 to the minute, the cardiac rhythm being loud and distinct with a markedly accentuated second sound at the base. During the last hour of life the pulse could not be felt at the wrist, and the heart's condition was not investigated. This pulse was certainly very remarkable, and no other case has come under the notice of the writer in which death occurred from the complication under consideration without the pulse rising for some days previously, and finally becoming uncountable at from 140 to 160 to the minute. As there was no other clinical symptom to separate this case from others of the same class, the writer concludes that here also there was some condition of the

vascular system which was not usual. At the post-mortem examination it was found that the muscle of the heart was remarkably firm and healthy, and that the wall of the left ventricle was hypertrophied to quite twice the ordinary thickness, while its cavity was not enlarged and the valves were in every respect normal. The evidence of such cases seems strongly in favour of the view that if the heart be sufficiently powerful it will not beat rapidly because of the existence of a simple inflammation, unless this be of the most intense character or complicated by other conditions which also add to the work of the cardiac muscle. From the above it may be deduced that the cause of the rapid pulse in inflammatory fever, and in the other conditions which have been under consideration, is *a weakness of the heart relatively to the amount of opposition to the blood-flow induced by the contraction of the arteries throughout the body.*

Physiological Changes illustrating the Effects of a Strong Heart on the Pulse Rate in Traumatic Fever.

It may be stated as a clinical fact that not only when individual cases, such as those given, are considered, but also when classes of cases are taken note of, in healthy subjects with healthy

wounds, the pulse changes in traumatic fever are less marked, longer delayed, and less easily brought about, according as the strength of the cardiac muscle is greater relatively to the opposition to the blood-stream in the vessels.

For instance, in healthy subjects who have reached middle life, and more markedly at a greater age,* it seems to be an indisputable clinical fact that the convalescence from such an operation as ovariotomy, when there is no complication, is characterised by less cardiac disturbance than is found in younger subjects, even although in each the ultimate result, as regards absence of suppuration and rapidity and completeness of healing, is apparently the same, and although both classes seem in equally good health.

It appears probable that, as a class, those who have reached or passed middle life have relatively stronger and more capable hearts than those who are younger. The vascular system is one of the last to become fully developed and consolidated; and the longer life continues, the greater seems to be the advantage gained in this respect. Sir George Humphry wrote† of the post-mortem conditions found in centenarians;—"It is not a little remarkable that, whereas the greatest appreciable wast-

* Sir George Humphry, "Remarks on the Repair of Wounds and Fractures in Aged Persons," 'Brit. Med. Journ.,' July 12th, 1884.

† 'Brit. Med. Journ.,' 1887, vol. i, p. 612.

ing is in the spleen and other organs which have relation to blood-making, the greatest increase is in the heart." He attributes the increase in part to accumulation of fat, to senile thickening, and other changes, but also in part to hypertrophy of muscular structure. Balfour says, "In those that live to great ages it (the heart) is mostly stronger and fitter for the discharge of its duties than at an earlier period.* Thus we may explain the fact that a man in the prime of life, and, in many cases, in healthy old age, can perform without any cardiac or respiratory difficulty an amount of work which, if undertaken by a youth, would inevitably cause signs of distress.

This does not imply greater agility or muscular power in the older man. In agility the young man usually excels, and in muscular power he may excel at a time when we know that his development is not fully matured. It is the power of doing continuous hard work—the staying power—that reaches its highest efficiency only when the development of the heart is complete.

This is also seen in the horse. In his second or third year that animal displays his utmost speed, and the best sprint-racers are about this age. It is not till he is about five or six years old, however, that the horse is fully developed,

* Geo. W. Balfour, 'Edin. Med. Journ.,' February, 1888, p. 682.

and acquires his greatest power for steeplechasing or other prolonged work, and it is well known that when a particularly long journey is to be done an aged horse is usually selected. The pace may be slower, but the old one can often stay the distance when a young horse cannot.

There is recorded, in the case of a dog, a remarkable example of unusual cardiac development combined with great endurance and speed. Master M'Grath, a celebrated greyhound, won the Waterloo Cup, which is the most important coursing event of the year, in 1868, in 1869, and in 1871. He died after a short illness on December 24th, 1871. Owing to a suspicion that the dog might have been poisoned, Professor Haughton, M.D., F.R.S., and secretary to the Royal Zoological Society of Ireland, made a post-mortem examination. There was double pneumonia, and both lungs "were occupied extensively with tuberculous deposits." The heart was "enormously hypertrophied, but in other respects healthy." Professor Haughton considered that "the hypertrophied condition of the heart caused a state of health bordering on disease, and liable to pass into it on the occurrence of any disturbance of the equilibrium of respiration and circulation."* However that may be, the association of an enormously hypertrophied

* The 'Field,' December 30th, 1871.

heart with a pre-eminence in the stoutness and swiftness which are required in coursing lends support to the view that the more powerful the cardiac muscle, the more easily will the animal perform feats of strength and endurance.

In like manner it may be argued that the fully developed and relatively stronger heart of middle and advanced age is able to perform the extra work necessitated by an attack of fever with little or no difficulty, and therefore with little or no acceleration of its action ; whereas, in a youth, and still more in a child, the immaturely developed and relatively feeble cardiac muscle must exert its utmost power to overcome the resistance caused by the febrile state, and a rapid pulse is easily induced.

In the child and in the youth, and until middle life, there is also a greater contractile power in the vessels than there is in old age. A greater constriction of the vessels is, therefore, apt to be induced at the same time that the heart is less capable of overcoming opposition. On the other hand, when a man has passed his prime the muscular tissue of the vessels is less active, and as age advances may become atheromatous. These conditions prevent any very powerful contraction of the arterial walls, and any very great increase of the heart's work from this cause, in the aged.

Of course it is not every individual who has

reached or passed middle life who can safely and easily go through the perils of a severe operation. There are exceptions to the rule that operations are well borne by elderly people. But a study of these exceptions frequently tends to throw into prominence the importance of the beneficial influence of a relatively strong cardiac muscle on traumatic fever. Investigation will show that in many cases in which older subjects exhibit signs of cardiac debility after uncomplicated operations there is distinct cardiac disease, or evidence of previous weakness of the heart muscle.

Sometimes both the power of the heart and the contraction of the vessels are unusually well marked. In the case of a woman twenty-five years of age, on whom a simple ovariotomy was performed, the heart seemed so strong and healthy on examination beforehand, that during the operation no anxiety was felt about her general condition. After the operation was finished, the patient had an extremely feeble pulse, felt with difficulty in the radial artery. On listening to the heart, however, very loud distinct heart sounds were heard. Apparently the cardiac muscle was contracting with great force, but it was beating only forty-eight times to the minute. It would seem that there was in this case an intense contraction of the arteries, while the heart was acting strongly and, in

accordance with Marey's law, slowly; there was the intense reflex constriction of the arteries which is common in youth, associated with the powerful cardiac action which is more usually found in later life.

The Condition of the Vascular System after Parturition.

The condition of a woman after parturition is in many respects comparable to that found after an injury in which drainage is free. In both cases there is a solution of continuity, and the feverishness which occurs in the lying-in state is similar in origin to that which follows an operation. It is a characteristic of the fever of childbed that the pulse is usually slow, often remarkably so, and is in this respect in strong contrast to the amount of rise of temperature.* This comparative slowness of pulse has been attributed by Spiegelberg to "the mental and physical rest of the puerperal woman, as well as a certain impoverishment of the blood due to its abundant losses while but a sparing amount of nourishment is taken, . . . perhaps also to some temporary disturbance of the nervous mechanism caused by

* 'Otto Spiegelberg's Midwifery,' translated by J. Hurry, vol. i, p. 289; 'Playfair's Midwifery,' vol. ii, p. 247.

the impoverishment."* The slow pulse has also been attributed to an increase of blood-pressure.† But "according to the most recent observations (Fritsch, Löhlein, Meyburg), the arterial tension is not increased."‡ Increased arterial tension, therefore, cannot be the cause of the slow pulse. On the other hand, the most complete mental and physical rest may be present without producing any unusual slowness of cardiac action, while impoverishment of the blood is more commonly associated with a rapidly beating and excited heart, and seems rather to aid in explaining the quick pulse which is so easily induced by complications during the puerperal state than the slow heart action of an ordinary case.

It has been shown above that a relatively slow pulse is sometimes found during convalescence from an operation, and that in such cases there is frequently evidence of an unusually powerful heart action. In women after delivery, when this slow heart action is usual, there is, as a rule, and in normal conditions always, an excess of cardiac power from a temporary hypertrophy of the left ventricle. This hypertrophy appears to be a physiological result of the extra work gradually

* 'Otto Spiegelberg's Midwifery,' translated by J. Hurry, vol. i, p. 289.

† 'Blot. Arch. Gen. de Med.,' 1864. Quoted by Playfair, 'Midwifery,' vol. ii, p. 247.

‡ Spiegelberg, loc. cit.

thrown on the heart as the uterus enlarges. On the delivery of the child the work of the heart is greatly diminished, and its hypertrophy gradually passes off; but there remains for a time an excess of cardiac power relatively to the amount of work required of the heart by the slight febrile reaction which usually occurs during convalescence. It is to be noted that the "retardation of the pulse is more frequently observed in multiparæ than in primiparæ,"* while it is in multiparæ that the cardiac hypertrophy is most marked.†

The well-known danger of heart disease in a pregnant woman is also evidence of the usefulness of a strong cardiac force in counteracting the dangers to which the lying-in woman is exposed; and those forms of cardiac mischief which give rise to most risk in pregnancy, namely, mitral stenosis and aortic incompetence,‡ are also those which interfere most with the propelling power of the heart.

The conditions which exist during pregnancy suggest that, if the hypertrophy of the heart in this state be a purely physiological change provided to meet the extra work caused by the enlarging uterus and child, a similar compensa-

* Spiegelberg, loc. cit.

† 'Playfair's Midwifery,' vol. i, p. 139.

‡ Ibid., vol. i, p. 243.

tory change may occur in other cases. For instance, an intra-abdominal tumour, if of any size, must throw a great deal of extra work on the heart. This may be brought about as gradually as in pregnancy, and might be expected, as in that condition, to produce a cardiac hypertrophy. The writer has seen two cases* in which a post-mortem examination proved the existence of such a condition; but there is much clinical evidence that this does frequently occur, and, reasoning from analogy alone, we might attribute to an hypertrophy of the cardiac muscle the truly marvellous manner in which patients sometimes recover after severe operations for the removal of abdominal tumours.

It is obvious that if such a salutary provision for convalescence takes place, those cases in which the change in the heart muscle is most marked must have the best chance of recovering from any surgical proceeding, so that post-mortem evidence in support of the hypothesis would necessarily be rare.

These remarks apply also to operations for long-standing inflammatory disease. It may often be observed that in cases of the most extreme exhaustion—from joint disease, for instance—after the removal of the diseased part by amputation, the patient immediately and rapidly proceeds to

* See p. 55, *supra*.

recover. There can be no doubt that the mortality is far greater in cases when an operation is performed after an injury than when it is necessitated by disease. According to the evidence given above concerning the effect of shock and inflammation on the vessels, and the importance of a relatively strong cardiac muscle, there is in the heart's condition a full and adequate explanation of this greater mortality in primary operations. A prolonged inflammation must throw much extra work on the heart, so that the preparation of this organ for convalescence in a case of operation for disease of a chronic inflammatory nature may be compared to a course of training, or to the physiological preparation which takes place during pregnancy; whereas, in a case of injury, the heart is called upon suddenly to exert itself, perhaps to its utmost power, by the shock of an accident. It is again severely strained during the operation, and is afterwards required to undergo a period of hard work during the continuance of the traumatic fever. There should be no greater wonder at a frequent failure under these circumstances than there is cause for surprise that a man whose habits and occupation are sedentary should break down if he begin a walking tour by doing thirty or forty miles on the first day.

Conclusions as to the Causes of the Various Pulse Conditions found in Fever.

The evidence seems very strong that in traumatic fever, in shock, and in abdominal distension following a laparotomy, the small feeble pulse, whether slow or fast, is in no sense due to cardiac weakness, is not a condition beginning at the heart, but is an immediate effect of the contraction of the vessels. As the vessels contract, the work of the heart is constantly added to. The excess of work thus thrown on the cardiac muscle is the cause of the quickening of the pulse rate, which only occurs when the reserve energy of the heart is not equal to carrying on the circulation without difficulty. Thus in simple traumatic fever, in shock, in intestinal distension following laparotomy, and during convalescence after childbearing, the vascular system may show any condition in a series, one extreme of which is characterised by such a powerful state of the heart relatively to the obstruction in the arteries that its action does not become more rapid, or may even, in accordance with Marey's law, become slower than usual, while the volume of the pulse at the wrist is more or less full and bounding. As the contraction of the arteries becomes greater, and extends to larger vessels,

the pulse at the wrist becomes small, weak, and compressible. If the heart be sufficiently strong its action remains slow. In that case a full firm impulse in the large vessels, and loud distinct cardiac sounds with an accentuated second sound at the base, are easily detected. If still greater contraction of the vessels takes place, we have the other extreme of these conditions, in which the heart is relatively very feeble. Then its pace increases progressively until the beats may be scarcely countable, while the pulse at the wrist becomes indistinct and running, or may disappear altogether. As the pace quickens the forcible impulse in the large vessels and the loud definite cardiac sounds become less evident.

Distinct indications of these conditions will not be made out in all, or even in most cases. For instance, a slow pulse continuing for days when the temperature and other signs indicate considerable fever, is a condition only met with occasionally after operations, though commonly observed in lying-in women. The whole series of vascular changes, from the slow, full pulse, to the absolute absence of pulse at the wrist while the heart is beating 160 or more to the minute, may be run through, however, in any given case, when the conditions are suitable for their production.

A rapid pulse which is almost imperceptible at the wrist has usually been attributed to cardiac

weakness; but as the arterial contraction increases, the work of the heart is greatly augmented. Nevertheless the heart goes on contracting and sending out all the blood which reaches it. Hence, when the arteries are unusually contracted, during an operation, and during the traumatic fever which follows, the cardiac muscle is really doing a very much larger amount of work than usual. If, during convalescence, abdominal distension should come on, the strain on the heart is still further added to and prolonged. The pulse may gradually increase up to 150 or 160 to the minute, while after the pulse ceases to be perceptible at the wrist the cardiac muscle goes on beating at this pace, or faster, for some time. This happens, as a rule, in the cases of death from ileus and pseudo-ileus which have been described.

It only requires to be pointed out to be accepted that a heart which can do all this cannot rationally be described as a feeble organ. To attribute the conditions found in these cases to cardiac debility is therefore an erroneous and unscientific method of stating the facts. Instead of attributing the small, feeble pulse of fever and of shock to cardiac weakness, we should rather regard the contraction of the arteries as the cause of this phenomenon.*

* Since the foregoing was written, Professor Roy and Mr.

The Effects of the Vascular Changes brought about by a Traumatic Fever.

Although the character of the pulse at the wrist depends on the state of the vessels more than on the strength of the heart, it may be said that cardiac weakness and cardiac overwork come to very much the same thing. Apart, however, from the desirability of accuracy in description, and in our ideas of any pathological or physiological condition, there is a great deal of evidence that neither weakness nor overwork of the heart is the cause of death in the cases under consideration. There is reason to believe that death from traumatic fever, from shock, or from pseudo-ileus following laparatomy, usually begins elsewhere, and not at the heart. In cases of pseudo-ileus following an abdominal section the evidence to this effect is most easily

Adami have published a paper on "The Physiology and Pathology of the Mammalian Heart," in which they prove, by exact experimental methods, that "there always is an increase in force of the ventricular contractions as a result of stimulation of certain sensory nerves, yet that this increase in force seldom, if ever, exactly counterbalances the increased resistance to contraction which results from rise of pressure in the systemic vessels, and that sometimes the increased force of contraction more than counterbalances the increased resistance, while in other cases the increase in force does not suffice to do so."—'Philosophical Transactions,' vol. clxxxiii (1892), B, pp. 256, 257.

made out, because in these cases a contraction of the vessels is induced while the inflammatory conditions are becoming less marked, and therefore the consequences of contraction of the arteries are to some extent separated from the other results of the inflammation.

Olshausen's and Verchère's Explanation of the Symptoms and of the Cause of Death in a Case of Pseudo-ileus following Abdominal Section.

After an abdominal section, if the temperature and pulse rise for the first two or three days, and then fall well down towards the normal, as if a healthy physiological recovery were about to take place, and if, during this fall of temperature and pulse, retention of flatus in the alimentary canal occurs and abdominal distension becomes gradually more obvious, the patient is certainly suffering from obstruction of the bowels or from a pseudo-ileus. A feeling of fulness and oppression of the chest ensues, and is followed by nausea and vomiting. At first, the vomit consists of clear serous-looking fluid mixed with anything the patient may have swallowed. It soon becomes bilious, and much yellow matter may be expelled. Later, the colour becomes greenish, and gradually changes until the vomit consists of a dark green or black fluid with a sour acrid

smell. This is thrown out of the stomach in large quantities at intervals, or in small quantities almost constantly.

As the distension of the abdomen increases, sooner or later a rise of temperature and pulse ensues. These, however, do not by any means rise and fall together. On the contrary, the most marked contrasts in the course of the temperature and pulse curves may be observed in the cases under consideration. When the bowels distend and the temperature falls about the third day, the pulse not infrequently remains at its high level. Or the pulse may increase in frequency while the temperature remains steady or falls. For example, in one case, just as distension of the abdomen was beginning, at 8 o'clock a.m., the temperature was 100·2° F. in the vagina; the pulse was 100. An hour and a half later the pulse was 120, the temperature 100·4° F. At 1.30 p.m. on the same day the pulse was 130, while the temperature, having been 100·6° F. in the interval, was again 100·4° F. That is to say, the temperature was very nearly steady for five and a half hours, while the pulse rose from 100 to 130. In another case, sixty hours after an abdominal section had been performed, there was great distension of the abdomen with retention of flatus, and frequent vomiting for some hours previously. At this time the temperature was

102° F., and it remained steady for the next eight hours. During that period there was no vomiting, and much flatus escaped from the rectum when a tube was passed through the anus at frequent intervals. The distension of the abdomen did not diminish, however, and the pulse rose steadily from 112 to 120. At the end of the eight hours flatus again ceased to pass from the anus, and vomiting recommenced. The temperature then fell to 101·4° F., and remained at that point for four hours, the pulse during this period rising to 140. Thus in twelve hours the pulse rose from 112 to 140, the temperature being three-fifths of a degree lower in the last four than in the first eight hours of this time. In both of these cases a distinct obstruction of the bowels was found after death.

The period at which the rise of temperature takes place is also very uncertain, but a rise invariably occurs, sometimes running up rapidly to 104° or 105° F. just before death.

In Olshausen's and Verchère's papers on death from paralysis of the bowels during convalescence from a laparotomy, the fatality is attributed to the absorption of poisonous substances from the alimentary canal. Verchère gives to the whole process the name of *septicémie intestino-peritonéale*. He says ;—" Le diagnostic est évident dans la plupart des cas et il était nécessaire de distinguer

cette entité morbide de la péritonite, avec laquelle elle a toujours été confondue." And again ;—" Les cavités abdominales ne suppurent plus comme autrefois, et si les malades succombent, ce n'est pas, comme le redoutaient les premiers chirurgiens qui ont tenté la laparotomie, par suppuration, mais par un autre mécanisme, par la septicémie telle que nous l'avons décrite." * The explanation is that given by most recent writers of the well-known fact that during any inflammation, in convalescence from any great operation for instance, if the bowels become constipated, the temperature and pulse are very apt to rise, but usually fall at once if the bowels act freely. That septicæmia is produced by the absorption of poisonous matter from the bowel is not, however, a satisfactory explanation of the signs and symptoms found in the cases under discussion, and its occurrence is quite unproved. It is well known that obstruction of the bowels uncomplicated by inflammatory mischief may be absolute, and yet the patient may continue to exist for many days, even for weeks, death being eventually due to a gradual exhaustion of the vital powers or to some accidental complication. But if obstruction of the intestine should become absolute within a few days after an abdominal section, death certainly follows not later than

* 'Revue de Chirurgie,' July, 1888.

three days after progressive symptoms of obstruction become well marked. The passage of flatus from the anus, a movement of the bowels, or the removal of a large quantity of fluid from the stomach by means of the œsophageal tube will alleviate the symptoms, and may delay the result. A marked temporary amelioration of symptoms may be brought about even by such a severe proceeding as opening the abdomen, incising the bowel, and so emptying it of its contents. But unless the obstructive condition be removed, death is inevitable. On the other hand, a patient may be apparently dying from pseudo-ileus, yet, if the abdominal distension be permanently relieved without the infliction of further injury, recovery may take place when all hope seems vain. The phenomena induced are therefore clearly due to the combination of the effects of the obstruction of the bowels with those of the inflammation caused by the operation. Conversely, it is found that all cases of rapidly fatal obstruction of the bowels are complicated by some inflammatory mischief, injury, or strangulation of tissue.

According to Verchère, "cette distension et aussi l'état particulier dans lequel se trouvent les parois intestinales permettront une sorte de filtration à travers les tuniques intestinales : de la un épanchement dans la séreuse abdominale ou de liquide septique ou de gaz putride. Ce liquide,

ce gaz sont chargés de micro-organismes qui trouvent un lieu d'absorption favorable dans le péritoine lui-meme, et la septicémie commence.

"MM. Verneuil et Nepveu ont pu saisir cette filtration sur le fait, et ont publié leur recherches à cet égard. Si l'on vient à examiner le liquide contenu dans un sac herniaire, surtout si celui-ci a été malaxé par le taxis, on trouve en quantité considérable des micro-organismes ; vient-on à réduire dans l'abdomen ce liquide septique, on est exposé à voir survenir tous les phénomènes graves de la septicémie peritonéale."

It is, however, to be noted that Verchère includes in his paper the consideration of a large variety of conditions, namely, accidental injuries to the abdomen, and operations for herniæ of all kinds, as well as laparotomies for various objects. He slumps together for the purposes of scientific evidence all these cases, whether the gut has been left without damage, or whether it has been seriously injured. He says, "À côté des plaies viscérales de l'abdomen, il faut placer sur le même rang comme cause de septicémie les plaies simples de l'abdomen. Ce sont les laparotomies, les ovariotomies et hystérectomies abdominales qui donnent le plus fort contingent de mort par la septicémie que nous étudions."

To embrace the results of such a wide range of observations, and to endeavour to reduce the

pathology of so many varying conditions to one standard, tends rather to confuse than to elucidate our ideas of the pathological changes under consideration. When an operation is performed for hernia, and a congested, much manipulated, almost sloughing piece of bowel is returned into the abdomen, there is little resemblance to the conditions which exist when death follows on an operation for strangulated hernia in which the promptitude of the surgeon has enabled him to return the gut before it has suffered any material damage. Still less can cases of severe contusion or laceration of the bowel be compared to cases of laparotomy, in which no injury whatever has been done to the intestine beyond its exposure and manipulation with the hands and with the sponges. Yet these cases do sometimes die with all the symptoms of a pseudo-ileus. Indeed, Verchère distinctly says that the "strongest contingent" of deaths from this cause is found in these simple cases.

Verchère and Olshausen have separated cases of death due to pseudo-ileus from cases of death due to peritonitis; but they have still confounded them, alike in their clinical and pathological aspects. They have made up their clinical picture from a consideration of both simple and complex cases. Their pathology is founded entirely on the conditions observed in complicated cases only.

Verchère's clinical picture, as a picture of a pseudo-ileus following laparotomy, is certainly incorrect in matters of fact. He says, "En résumé, les symptômes sont de deux ordres : des symptômes locaux et des symptômes généraux. Les symptômes locaux sont : le ballonnement du ventre, la constipation, la rétention des gaz, *l'indolescence absolue de l'abdomen ;* les symptômes généraux : la rapidité et la petitesse du pouls accompagnée d'une température basse, ou normale, jusqu'aux derniers moments, où elle monte subitement à 40° ; des vomissements bilieux, puis fécaloïdes ; le facies abdominal, un affaiblissement progressif, puis la mort."

There is no *facies abdominalis* in a typical case of death from obstruction of the bowels, after a laparotomy which, but for the obstruction, has shown signs that recovery would have ensued. Sir Spencer Wells, in describing one of his early cases, says that "the patient became gradually weaker, and although quite sensible, cheerful, and hopeful till half an hour before death, she sank in the afternoon, 102 hours after the operation."* The account of this case strongly suggests that death must have been due to a pseudo-ileus, and not to a peritonitis, as stated. However this may be, the sensible, cheerful, hopeful condition is frequent in cases of pseudo-ileus and obstruction. Even

* Wells, 'On Diseases of the Ovaries,' Case 10.

if the patient should realise that death is imminent, there is no *facies abdominalis*. In a paper already referred to,* this sign was given a most important place as an indication that septicæmia is present. Of course, in a case of septic peritonitis, unless the inflammation be defined by adhesions, and of limited extent, all the other signs given above as due to paralysis of the bowel or to obstruction must also exist; for a severe inflammation of the peritoneum covering the gut necessarily induces paralysis of the affected part.

As regards the pulse and temperature, a feeble rapid pulse throughout, and a normal or subnormal temperature till the last moments, are by no means the conditions found in a typical case of pseudo-ileus after laparotomy. The pulse and temperature invariably follow the same course as they naturally would after any operation, until the distension of the bowel becomes so great as to induce symptoms of intestinal obstruction.

In making the vomiting a general symptom, Verchère evidently means to attribute it to the septic condition which he believes to exist. He says, however, that in the cases he relates "c'est la description classique des symptômes accompagnant une hernie étranglée, c'est la description

* "The Condition and Management of the Intestines after Abdominal Section," Malcolm, 'Med.-Chir. Transactions,' vol. lxxi

de toute occlusion intestinale, et aussi des pseudo-étranglements bien connus depuis les travaux de Henrot." Again we observe the absence of any differentiation between obstruction of the bowels, and obstruction complicated by an inflammatory condition. Surely in the case of an uncomplicated obstruction of the bowels the vomiting is an immediate result of the inability of the gut to force its contents downwards. It seems evident that both in simple obstruction and in pseudo-ileus after laparotomy the vomiting is a direct consequence of the constipation and retention of gases, which are undoubtedly due to local changes in the gut. In health, when digestion in the stomach is complete, its contents are sent downwards into the small intestine through the duodenum. If, however, an obstruction exist at the duodenum, vomiting results, which is frequent and violent when food is taken, less obtrusive when food by the mouth is withheld. But it recurs at intervals, even though no food be allowed; for as fluid collects in the stomach, if it cannot pass through the duodenum, it will certainly cause nausea and vomiting. An obstruction at any point of the alimentary canal first stimulates peristalsis, and increases the flow of secretion into the bowel above the point of occlusion. If persistent, the obstruction afterwards brings about a dilatation of the gut from accumulation of its contents. This dilata-

tion spreads upwards, and sooner or later vomiting follows. That this is, in great part at least, brought about by the accumulation of secretions and gases is evident from the fact that if a large quantity of fluid be vomited at a time, there is a lengthened period before the next act of vomiting occurs, and if the stomach be thoroughly emptied artificially, there is a still longer rest. On the other hand, if very little comes at a time, the vomiting may be almost continuous. Without doubt, however, reflex nervous changes also conduce to bring about vomiting as an effect of obstruction, for a loaded rectum may cause sickness and vomiting although there may be no general abdominal distension.

When an obstruction occurs after an abdominal section the amount of fluid vomited is often very great, and is probably (*cæteris paribus*) greater than in simple obstruction occurring in the absence of an inflammatory process. A very dark colour of the vomited matter is also more constant after operations than in uncomplicated obstruction of the bowels. From the fluid which accumulates in the alimentary canal, it is assumed by Verchère, Olshausen, and many others that a poisonous matter is absorbed which is the cause of the symptoms, and of death if death ensue. Nevertheless it is easy to observe in these cases that, while the obstructive condition

continues, large quantities of fluid are thrown into the alimentary canal, and are in great part ejected by the act of vomiting. During the continuance of this process the condition of the patient grows steadily worse. If, on the other hand, as not infrequently happens, the obstructive condition be suddenly removed by nature or by art, so that the flatus passes downwards, the vomiting immediately ceases. In that case it is seldom that a loose motion of the bowels follows, unless the cure be due to purgation. The bowels do not usually move for some days, and when they do, the stool is not specially soft. On the contrary, it is often very hard. Hence it may be inferred that, when the vomiting ceases, the liquid contents of the bowel are absorbed in considerable quantity. But the escape of flatus through the anus and cessation of vomiting in these cases are invariably accompanied by a marked improvement of the general condition. To put the matter shortly :— on the one hand, when there is every evidence that fluid is being thrown out of the tissues and ejected from the system by vomiting, the patient steadily loses ground ; on the other hand, when it is extremely probable that a considerable quantity of this same fluid is being absorbed, the general condition rapidly improves. Nevertheless we are asked to believe that the adverse symptoms and death in these cases are due

to the absorption of a poison generated in this fluid.

The evidence to be found in these conditions is very inadequate to support such a conclusion. The vomiting in cases of pseudo-ileus following a laparotomy seems to be brought about as it is in a simple obstruction of the bowels, namely, by a physiological mechanism set in action by the abnormal conditions which arise when the peristalsis is unable to propel downwards the intestinal contents. The greater quantity and the character of the vomit are fully explained by the excessive destruction of blood which takes place during the inflammatory process, and by the consequent increased secretion and altered character of the bile.*

The Effect of a General Contraction of the Vessels on the Tissues.

The evidence of the existence of any form of poisoning in these cases being thus defective, the question arises whether there is any other possible explanation of the signs and symptoms observed. Another, and a more rational interpretation of the processes which precede death may be given.

We have seen that with the increase of the abdominal distension there is a progressive narrowing of the peripheral arteries; that this contraction may spread to, and beyond, vessels of the

* Vide p. 101, *infra*.

size of the radial; and that the effect of it is to raise the blood-pressure in the larger arteries, but to diminish pressure in the small arteries, through which the blood may even cease to flow.

Whether the contraction of the peripheral arteries be at first general or be confined to certain areas, if it become sufficiently intense, either the affected areas must be deprived altogether of blood, or the regulating power of the vaso-motor system must come into play, and contract the arteries of every other part of the body, or, it may be, first of one area and then of another until all are involved. Evidence can be adduced that this contraction of the vessels has a marked effect both on the tissues of the body and on the blood itself.

First as regards the tissues:—In the cases under consideration, the urine invariably diminishes in quantity and becomes of lower specific gravity as the symptoms of obstruction progress. These changes may be observed twenty-four hours before death, and gradually become more obvious until urine ceases to enter the bladder, often for many hours. Complete suppression of urine has been observed for ten hours before death from pseudo-ileus, but in no case of this kind has the writer detected albuminuria. If the obstruction of the bowels be relieved sufficiently soon the secretion of urine returns, and the kidneys do not show any further sign of want of power.

In health the amount of urine depends very closely upon the difference between the pressure of the blood in the glomeruli and the pressure within the renal tubules. When the pressure within the tubules is increased, as in cases of obstructive unilateral hydronephrosis, the urine secreted by the hydronephrotic kidney is not only diminished in quantity, but, in every case observed by the writer, he has found it also of very low specific gravity, much lower than that secreted by the other kidney. Diminished pressure in the blood-vessels of the glomeruli must have the same effect as increased pressure in the tubules, so far as the relative pressure of the two is concerned. Hence in diminished blood-pressure within the glomeruli we have a sufficient explanation of the state of the urine in the cases under consideration. Moreover, diminution of the urine and lowering of its specific gravity do not occur in these cases until there is a marked smallness of the radial pulse. It seems, therefore, that the failure of renal action must be due to a diminished pressure in the renal vessels from a too great contraction taking place in them.

About the time that the urine begins to diminish, or soon after this occurs, there is another obvious sign of increasing contraction of the arteries. The extremities gradually assume a death-like coldness, which extends, but less

markedly, to the surface generally. This occurs even though the rectal temperature may be rapidly rising, perhaps to a very great height. The coldness may be observed five or six hours, and sometimes much longer, before death, and it is always accompanied by a small pulse.

The suppression of all manifestations of mental activity is the next evident change. As in Sir Spencer Wells' case already referred to, the patient is usually "quite sensible, cheerful, and hopeful, till half an hour before death." About this time the condition passes into one of coma,—a few rambling remarks, a complaint of feeling giddy, or a slight restlessness being the only indications that such a change is taking place. The exclusion of blood from the organs of thought and from the motor areas is a sufficient explanation of this comatose condition; and, as we have seen that the circulation is gradually being shut off from the tissues and organs by the progressive contraction of the blood-vessels, it is rational to assume that the unconsciousness is due to this action affecting the vessels of those parts in which the intellectual processes are carried on.

When such large and important areas have their blood-supply arrested, it is probable that the abnormal reflex stimulus to contraction of other vessels must greatly increase. The vital centres in the medulla which regulate the action of the

lungs and heart are at length affected, and when the exercise of the function of these centres ceases, life itself is at an end. Death is due to an excessive contraction of the arteries, brought about through a reflex *physiological* mechanism, by the want of fresh blood in the inflamed area, and by the opposition to the flow of blood through the vessels of the distended intestine. Evidence of the continuance of some at least of the functions of the medulla till the very last moments of life is found in the fact that failure of the respiration only takes place a few minutes before death. The breathing which has been slow and stertorous then becomes irregular, and finally there is but a spasmodic inspiratory effort recurring at intervals, which gradually lengthen till the last inspiratory gasp occurs, often after the patient is supposed to have completely expired.

During this final period, when the patient is virtually, but not actually dead, if the heart be listened to, doubt must, in many cases, at once arise as to the correctness of any explanation of the conditions observed which assumes that cardiac failure is an important element in their causation. At this time, although the pulse may be absolutely imperceptible at the wrist, the heart-beat may be heard, in some cases at least, clear and distinct, and with little or no irregularity, although the breathing is a mere involuntary

spasmodic contraction of the diaphragm occurring three or four times in the minute. Evidently a flow of blood through the heart goes on even then. There must, therefore, be certain vascular areas which are not, up to the very last, so contracted as to stop the circulation. This goes on until, and apparently even after, the vital centres in the medulla cease to act. Probably some blood passes from the arterial to the venous system through the more central and warmer parts of the body, and so gets back to the heart. Hence there is often congestion of the internal viscera found after death, as in some fatal cases of shock.

The signs and symptoms which have been described point to the conclusion that the condition of the vascular system in the cases under consideration is one of increasing arterial contraction, which, if its cause be sufficiently continuous and powerful, arrests the supply of blood to the various tissues, one vascular area after another being affected, until the medulla oblongata itself is deprived of nourishment, and the patient dies. Death is in these cases wholly dependent on the persistence of the cause, namely, the increasing abdominal distension, and is scarcely, if at all, influenced by the power of the heart. It would seem that when death results from an inflammatory process the heart is never stimulated to its greatest activity. It certainly never beats, in

consequence of an inflammation, at a rate of between two and three hundred to the minute—a pace which has been recorded by Bristowe * and West,† as induced by other conditions. Of course similar signs and symptoms may, to a certain extent, be produced by cardiac feebleness, and weakness of the heart facilitates their development, but the strongest heart cannot prevent death from pseudo-ileus after an abdominal section.

The advancing contraction of the arteries must have an important effect on the tissues in the neighbourhood of the inflamed area. It has been pointed out that the contraction of the arteries throughout the body, when considered in connection with the dilatation of the vessels in the area around the irritated part, yields a complete explanation of the determination of blood which occurs in inflammation. The area of dilated arterioles is, however, limited in extent. There is no evidence that the particular vessels leading from the heart to the inflamed area are dilated except in those terminal branches which are in the immediate neighbourhood of the irritation. If, therefore, the contraction of the vessels generally becomes so great as to affect the larger vessels which are well away from the local dilating influence of the injury, the blood may become shut off from the

* 'Brain,' vol. x.

† 'Brit. Med. Journ.,' 1890, vol. i, p. 605.

inflamed area as from any other. But a free flow of blood around the healing parts is essential to the completion of the process of resolution. If an obstruction occur to the flow of the blood in the veins leading from an inflamed part before resolution is complete, an unhealthy action is certain to be induced in the wound. On the other hand, if the force of the blood-current flowing towards an injured part be greatly weakened, an unhealthy process is then also apt to take place in the inflamed tissues. This may be observed when the access of blood to the part is greatly impeded, as in a very thin flap. Under such circumstances a dusky redness of the edges of the wound from passive congestion, and even a sloughy condition may result. Thus, whether the supply of blood to, or the flow of blood from a healing part be interfered with, a deficient nutrition of the tissues is brought about. A return of inflammatory action is apt to follow, and an inflammation thus induced tends to assume an unhealthy asthenic character.

In cases of abdominal section, when distension of the intestine occurs as the primary changes in the wounded tissues are subsiding, the healing process in the abdominal wall and within the abdominal cavity must be subjected to both of the vascular conditions which tend to produce unhealthy action. The direct pressure must

greatly interfere with the flow of blood from the part, while both the direct pressure and the reflex contraction of the arteries must hinder the flow of blood to it. In these cases, therefore, as abdominal distension increases, a return of inflammation in the divided tissues, and this of an unhealthy asthenic type, is sure sooner or later to take place. That the blood is, to a great extent at least, shut off from the inflamed part is proved by the fact that, though there is abundant clinical and post-mortem evidence of a return of inflammatory action in the wounded parts shortly before death, and undoubted evidence of intense contraction of the arteries elsewhere, there is never any sign of an active determination of blood to the inflamed area as death approaches. On the contrary, abundant post-mortem evidence of passive congestion is frequently found. It seems, therefore, certain that in these cases the contraction of the arteries shuts off the blood from the peripheral tissues which are undergoing the healing process, and that this is an important cause of the exacerbation of inflammatory action which occurs in the wounded parts before death.

In the cases of abdominal section in which the writer has ascribed the deaths to paralysis or obstruction of the bowels, it has been shown * that there is a time, after the symptoms of

* P. 38, *supra*.

obstruction are well developed, when no sign of peritonitis is found on reopening the abdomen. We have seen also that, as death approaches, there is reason to believe that the circulation goes on longer in the inner and warmer parts of the body, which are apt to show marked congestion after death. Thus, although the peritoneum may exhibit its usual smooth glistening surface, a deep blue-black colour of that membrane is often observed, showing clearly that a local dilatation of its vessels takes place, and that only a feeble blood-current passes through them. When such conditions exist, it is not to be wondered at that an asthenic inflammation, in a wound which implicates the peritoneum, is apt to spread and to induce diffuse peritonitis. The writer has repeatedly seen the signs of peritonitis well marked in the neighbourhood of the abdominal incision, but shading off, so that the peritoneum in the posterior and upper parts of the abdominal cavity was quite healthy in appearance. In two such cases there was a complete obstruction of the bowels, without any sign of inflammation of the peritoneum on the gut or adjacent to the gut at the point of obstruction. He has also noted that when death occurs very quickly after the rise of temperature commences, signs of peritonitis are frequently absent or slightly marked. On the other hand, the longer the patient lives after

the secondary rise of temperature begins, the more distinct are the evidences of spreading peritonitis which are found post mortem.

It is to be observed that the patient is really dying when the secondary inflammation in the wound comes on. This inflammation, and the peritonitis which usually spreads from it, are not the causes, but the consequences of the tympanites. Inflammation of the peritoneum is, in these cases, only an incident in the mode of death; and therefore, although it is almost invariably present, it cannot properly be called the cause of death. It cannot even be accurately described as the immediate cause of death, for some cases die without any sign of peritonitis being discoverable afterwards.

When the fresh burden of an unhealthy inflammation is thrown on the system, whether it becomes diffused over the peritoneum or not, death is inevitable and rapid. The secondary inflammation thus induced appears to be the cause of the rise of temperature—the ante-mortem rise—which invariably occurs in these cases. In this lies the explanation of the difference of the effects of the condition under consideration on the pulse and on the temperature. If the temperature does not rise till an extension of the inflammatory action is excited, and if this extension of the inflammatory action be due to the condition of the vessels, it is obvious that the

vascular changes must precede the rise of temperature. Of course when the inflammation does readvance, the pulse is still more affected.

In the foregoing considerations there is a full explanation of some cases of so-called "latent peritonitis." When a part is bruised so as to undergo extravasation of blood, there is not necessarily any consequent constitutional disturbance. But if a tight bandage be placed round a limb above such an injury so as to cause venous congestion, an unhealthy action will certainly be induced around the bruise. So, if the abdomen be injured, an unknown amount of damage of internal organs and of the wall of the intestine may result. Under treatment by rest and judicious dieting there may be no sign that any injury whatever has been done. But if a patient be allowed to take ordinary nourishment before the extravasations have been reabsorbed, the bowel may be unable to propel its contents downwards. Even with the most skilful treatment some distension from paralysis of the bowel may result, and may lead to a pseudo-ileus from the venous hyperæmia and extravasations into the wall of the gut, as described by Olshausen. When this distension of the bowel occurs, an unhealthy peritonitis may be induced in the damaged parts by interference with the circulation, just as a secondary inflammation is brought about by the

same cause in the healing parts in cases of pseudo-ileus after laparotomy. In a case of bruising causing paralysis of the bowel there would probably be no adverse symptom until distension of the gut occurred, which, under proper treatment, might not be for three or four days after the injury. Under such circumstances one would expect a normal temperature until inflammatory action had begun to advance, and then a rapid rise before death, the pulse being also normal until distension became marked.

In uncomplicated traumatic inflammation, when death results, one may trace all the signs of a gradual shutting off of the blood from the tissues as above described. In more acute cases these signs are not so easily detected and separated from each other as in cases of pseudo-ileus following laparotomy, because they develop more rapidly, and at the same time as the primary rise of temperature and pulse is taking place. Moreover, death from inflammation is very rare without the presence of some complication—most commonly septicæmia—which renders the symptoms less definite. With the cases of pseudo-ileus as a guide, it is, however, not difficult to detect, in death from acute inflammation, all the evidences of the vascular changes described. The small quickening pulse, the cold extremities, coma, and death, with the heart beating up to and almost

beyond the very last moment of life, may all be observed. In death from acute traumatic fever also, when there is no distension of the bowels, although there may be great determination of blood to the wounded part, this does not continue to the last, as it would do if the arteries leading to the part remained dilated. The tissues around the wound become dusky and livid, evidently from passive congestion. We must conclude, therefore, that in these cases also, as death approaches, the blood-supply to the inflamed area is diminished by contraction of the vessels leading thereto.

In the contraction of the arteries there is also a rational explanation of those cases in which death occurs from twelve to sixty or more hours after an operation, apparently from the severity of the procedure. Such cases are variously attributed to "shock" and "exhaustion." Neither of these terms, however, carries with it an altogether satisfactory explanation. The patients certainly rally to some extent, so that the term "shock" is not quite appropriate. On the other hand, the term "exhaustion" should be reserved for cases of death from a much more prolonged illness. In the cases under consideration the contraction of the vessels due to the shock does not relax before that due to the inflammatory changes comes on with great severity. The urinary secretion is never free; it may be com-

pletely suppressed from the first. The patient may not become properly warmed. Full mental activity is not restored. After a variable time death takes place, with all the evidences of intense contraction of the vessels. The fatal result depends on an excessive degree of injury as compared with the vital power of the patient and with the regulating force of the vaso-motor and thermal systems. Perhaps in these cases more than in any other the fatality may be attributed to uncomplicated traumatic fever. Such deaths only occur in very feeble individuals, or after the most severe operations. They must be carefully separated from those due to acute septicæmia, which they may closely resemble, but which may occur in the most healthy and after any operation, however slight.

Here it may be remarked that the vascular conditions in death from inflammation, as above related, have a close similarity to those ascribed by some to death from old age.

Bichat says that "in the death which is the effect of old age the whole of the functions cease, because they have been successively extinguished. The vital powers abandon each organ by degrees, digestion languishes. the secretions and absorptions are finished, the capillary circulation becomes embarrassed; lastly, the general circulation is suppressed. The heart is the *ultimum moriens*. Such, then, is the great difference which

distinguishes the death of the old man from that which is the effect of a sudden blow. In the one the powers of life begin to be extinguished in all the parts, and cease at the heart; the body dies from the circumference towards the centre: in the other life becomes extinct at the heart, and afterwards in the parts; the phenomena of death are seen extending themselves from the centre to the circumference."* If, however, the view of the pathology of death from inflammation described in these pages be the correct one, then death from inflammation is only a very rapid example of death beginning at the periphery and terminating at the centre. In respect of the vascular changes, death from inflammation is as much in contrast with death from syncope, due to cardiac weakness or disease, as death from senility can be. In inflammation the heart is the last part of the body to give up the struggle, as Bichat declares is the case in old age.

When the heart is so overworked as it is in a sthenic fever, cardiac failure and sudden death may be easily brought about by an imprudent or involuntary muscular effort. A fatal termination of a case may thus occur during the effort of defæcation or in the act of vomiting. The writer has seen one patient die from sudden cardiac failure due to choking during an attempt at deglu-

* Bichat's 'Recherches physiologiques sur la vie et la mort,' translated by F. Gold, p. 143.

tition. The patient was undoubtedly dying in the slow way which is common in cases of pseudo-ileus after laparotomy; and nothing could have been more marked than the contrast between the usual mode of death in these cases and the sudden cessation of respiratory and muscular effort, the immediate and absolute extinction of all physical and mental power, which was brought about by the violent struggle for breath.

The Effect of the Contraction of the Vessels on the Blood.

The effect on the blood itself of the existence of the febrile condition is great, and certain experimental and clinical facts are of importance as aids to our comprehension of the results produced. Constant interchanges go on between the fluids in the tissues and the fluids in the blood-vessels, and between the fluids in the tissues and the contents of the lymphatics, while the lymphatic system also communicates directly with the venous system. When a shrinkage of the vascular capacity occurs there must necessarily be a diminution of the amount of blood in the vessels, and the fluids of the body must therefore accumulate in increasing quantities in the tissues and lymphatics. Hence when the arteries contract in inflammation, in the absence of venous congestion, there must be much tension induced

within the tissues or in the lymphatics throughout the body. In a favorable case this tension is physiologically relieved by the action of the excretory glands, and especially of the sweat glands. It is to be noted that an extreme contraction of the arteries, or even the shutting off of the blood-supply altogether, will not prevent the action of these latter glands. The circulation through the superficial parts may be obviously deficient while sweating is profuse. The condition of shock shows this well. Again, in persons whose peripheral circulation is feeble, the cold hands and feet are often markedly moist with a clammy sweat. Profuse perspiration may also be observed in nervous conditions, such as great fear or anxiety, in which there is at least no reason to suppose that an increased flow of blood takes place to the skin, in which, on the contrary, pallor is often extreme. Moreover sweating is a symptom of profuse hæmorrhage, and the more rapidly the body is drained of blood the more excessive is the perspiration. Clinically, therefore, it can be shown that sweating does not depend on the amount of the supply of blood to the sweat glands. There is, moreover, absolutely conclusive experimental proof that sweating may occur independently of the condition of the circulation, for secretion of sweat in the feet of a cat may be induced by stimulating the medulla

oblongata three-quarters of an hour after death.* The sweating which invariably takes place if an inflammation pursue a healthy course would, therefore, seem to be a reflex effect of the excess of fluid in the lymphatics and in the tissues.

Sweating indirectly removes much of the fluid parts of the blood. There is no evidence, however, that the blood-corpuscles are carried off in this way. If only the fluid parts be got rid of, a very great increase of the number of red blood-corpuscles relatively to the plasma must result. This, however, is not found to be the case. Dr. Lockhart Gibson has recorded that "the white corpuscles always rise in number, and the red corpuscles always fall in number, for a day or two after operation; and that without any regular proportion to the amount of blood lost."† The red corpuscles must, therefore, be eliminated in some way. In health these corpuscles are being continually broken up, and are as steadily being formed; but the rate of formation and destruction varies. The changes are greater in youth than in old age, and are very active after food has been taken. During the febrile process also there is evidence that the red blood-corpuscles are destroyed in great numbers. The amount of this destruction at any time may be estimated by

* 'Stirling and Landois's Physiology,' 4th edit., p. 555.

† 'Journal of Anatomy and Physiology,' vol. xx, p. 133.

the quantity of bile pigments formed.* That there is not usually any very obvious sign of an excess of bile formation in an ordinary case of traumatic fever is no proof that such does not take place. "Normally a great part of the bile goes round in a circle from the liver into the duodenum, thence into the blood, so to the liver again, while another part is carried down by the contents of the intestines, and after becoming more or less altered passes out of the body with the fæces."† It is therefore difficult to estimate by direct observation the amount of bile formation, although bile sometimes passes in the stools in obviously excessive quantity during a fever. There can be no doubt, however, that an increased formation of bile or of certain constituents of the bile does occur during the febrile state. It has been shown that the formation of bile and of urea depends in large measure on the destruction of red blood-corpuscles, and that these excretions are therefore necessarily increased or diminished together.‡ In fever the urea excretion is greatly in excess, and the urine pigment derived from the hæmoglobin may be multiplied twentyfold.§

* Hunter, 'Brit. Med. Journ.,' August 3rd, 1889.

† Lauder Brunton, 'Disorders of Digestion,' p. 185.

‡ Noel Paton, 'Journal of Anatomy and Physiology,' vol. xx, p. 521.

§ Landois and Stirling, loc. cit., 4th edit., p. 413.

There is thus distinct evidence of increased destruction of blood-corpuscles during the febrile process.

In cases of obstruction of intestine or of pseudo-ileus after laparotomy, along with the progressive contraction of the blood-vessels, there is marked evidence of great destruction of blood-corpuscles before the secondary rise of temperature takes place. There is a very obvious diminution of excretion through the kidneys, amounting sometimes to complete suppression before death; but there is invariably a great and increasing quantity of indican in the urine, which may be observed before any signs of suppression are detected. The amount of pigment vomited is also very great, while after death the liver is dark coloured and the gall-bladder is distended with black bile. This altered bile is certainly produced in very large quantity, and we must conclude that there is a correspondingly great destruction of red corpuscles. It is further to be borne in mind that this excessive destruction of blood-corpuscles is, in these cases of pseudo-ileus, contemporaneous with the contraction of the arteries, and not with an increase of temperature. On the contrary, the temperature is, sometimes at least, falling when the vomiting of altered bile is becoming excessive.

In shock nothing is known, as far as the author

is aware, of any effects of the condition on the amount of bile and urea excreted; but convalescence from shock is often preceded by bilious vomiting, which may be due to increased secretion of bile and other fluids.

With the certainty that the blood-plasma is diminished, there is thus a considerable amount of evidence that the red corpuscles are also destroyed and excreted in conditions in which great contraction of the arteries occurs. Recently acquired information suggests the possibility that the various constituents of the blood may be broken up and excreted nearly in the proportions in which they exist in health. Dr. William Hunter has shown* that, although variations in the composition of the blood and in the percentage of its corpuscles do occur, and in certain diseases may be very great, yet many of the alterations of composition observed in the blood are "merely changes in virtue of its function as the carrying tissue of the body. . . . The most remarkable feature presented by the blood is not, as is usually supposed, its varying composition, but the remarkable power it possesses of maintaining a composition as rightly entitled to be termed stable as that of any other tissue of the body."

* Arras and Gale Lectures, 'Brit. Med. Journ.,' 1889, vol. ii, p. 116.

In normal conditions the destruction of blood-corpuscles is accompanied by a corresponding amount of blood formation, so that the blood remains nearly or quite constant in quality and quantity. Hence increased formation of blood would seem to be a physiological consequence of excessive destruction of blood. But while the capacity of the vascular system remains small, or is being reduced, increased blood formation can only lead to still greater blood destruction. The formation of such a complex entity as a blood-corpuscle must, however, make great demands on the supplies of nutriment, and therefore any great increase in the activity of blood formation is quite sufficient to account for the rapid wasting of the fatty tissues and skeletal muscles which undoubtedly takes place during fever.

The Temperature in Traumatic Fever.

There cannot be any doubt that the contraction of the arteries, to which so many of the symptoms that are observed in fever have been traced, may exist in a very marked degree without any elevation of temperature being found. Moreover, great disintegration of the blood may occur when the temperature is falling. It has been pointed out by Burdon Sanderson that "the

febrile augmentation of the urea discharge takes place immediately after subcutaneous injection of pus, *i. e.* at a time which precedes the elevation of temperature." * It is therefore certain that an excessive destruction of red blood-corpuscles may take place when there is no elevation of temperature, and consequently that the elevation of temperature in traumatic fever does not depend on the increased destruction of blood-corpuscles.

In seeking for the cause of the elevation of temperature in fever, while attributing the excess of excretion of bile, urea, and the like products to the unusually active destruction of blood-corpuscles necessitated by the advancing contraction of the arteries, it might be suggested that the rise of temperature is coincident with, and caused by, the compensatory increased formation of blood. But "the urea discharge remains excessive during the whole course of the fever." † Therefore an excessive destruction of blood must go on also during the whole process. The patient, however, towards the end of an uncomplicated traumatic fever is evidently gaining strength and blood. At this time not only must blood be formed to replace that which is being destroyed;

* Burdon Sanderson, "On the Process of Fever," 'Practitioner,' April, 1876, p. 7 of reprint.

† Burdon Sanderson, loc. cit., quoting Senator, 'Untersuchungen über den Fiebershaften Process, und seine Behandlung.'

but as the vessels relax, the increasing intra-vascular space must also be filled up. Hence blood formation must continue in excess, if indeed it does not become still more active, as resolution progresses. The temperature is, however, falling during this time, and therefore increased blood formation cannot be the cause of the elevation of temperature.

Nevertheless, the formation of blood-corpuscles in unusual numbers must involve a large amount of tissue change in many parts of the organism. Calorimetric experimental evidence shows that, while these processes of excessive blood formation and blood destruction, and the wasting of the tissues and their subsequent return to the normal are going on, there is an increased elimination of heat. The variations in the amount of heat development and heat elimination which take place in health are very great. The taking of food, starvation, active exercise, repose, the temperature of the surroundings, and other conditions, have a marked effect on the amount of heat developed and eliminated. But in health these conditions have little or no influence on the body temperature. Elevation of temperature, therefore, does not depend on increased production of heat. In fact, less heat is produced in fever on fever diet, than in health on full diet.*

* Wood, 'On Fever,' p. 239.

As already pointed out, an elevation of temperature may be brought about by the stimulation of a particular nervous tract in the brain substance, whereby, it is said, the thermogenetic function of the muscles is abnormally increased. But if an elevation of body temperature does not depend on an increased production of heat, then no such explanation of the thermogenetic, or rather heat-raising effect of stimulation of the particular region to the inner side of the corpus striatum is satisfactory. A more rational view of the thermal phenomena of fever is that elevation of temperature depends in no way on increased production of heat, but on something which disturbs the arrangement whereby heat production is counterbalanced by heat elimination. The mere stimulation of a thermogenetic tract would not be expected to cause a rise of body temperature whilst the rest of the heat-regulating mechanism remains intact. With this mechanism in a healthy condition, an extra development of heat should at once produce compensating thermolysis, as when food is taken and after violent exercise. Therefore, it may be argued that thermolysis must be interfered with at the same time that thermogenesis is stimulated, or the temperature will not rise.

Every assumption that the muscles are in a special manner the heat-producing tissues—that

they have a thermogenetic function peculiar to themselves, and that on the excessive exercise of this function the febrile condition depends, is open to the same objection Doubtless the changes which take place in fever, and which lead to marked wasting of the muscles, are associated with the development of heat in the muscles, and are not dependent on nor associated with the exercise of the contractile function of their tissue. It has been shown, however, that there is some reason to suppose that the wasting of the muscular tissue during the febrile process may be secondary to the efforts made by the organism to supply fresh blood in place of that which is destroyed. It might be argued from this that the blood-forming organs have the greatest claim to be described as specially fever-producing; but then the blood-forming function is also very active when fever is abating. There must be great difficulty in establishing a special claim to the title of thermogenetic for any tissue. Probably all the tissues are thermogenetic in proportion to their bulk and vascularity.

An explanation, which is believed to be new, of the rise of temperature in fever has suggested itself to the writer. It has been shown that while the inflammatory process exists, there is, in the affected part, a temporary and partial devitalisation of tissue, which is the "primary lesion in

inflammatory congestion." There is also evidence that, while the process of inflammation is advancing, an increasing area is subjected to this condition of diminished vital activity. It has been suggested that this local condition, although the parts are full of more or less stagnant blood, and are surrounded by a very active circulation, may transmit to the vaso-motor centre an indication that there is a want of fresh blood in the part. Exactly in the same way, although the parts are surrounded by an active circulation of blood above the normal body temperature, and although they are themselves actually above that temperature, yet in the process of dying or becoming partially devitalised, as the normal, local, chemical reactions cease, the nerves of the affected tissues must transmit to the central nervous system an indication of increasing physiological inactivity—of the approach of the coldness of death. This would be expected reflexly to stimulate heat production. But if the body temperature be raised, those tissues which eliminate heat—notably the skin, or some elements of the skin—will be reflexly stimulated to increased action. Thus the local condition tends to raise the temperature, while the heat-regulating mechanism endeavours to bring it to the normal degree. Consequently the temperature of the body in traumatic fever must depend at any particular moment on the

comparative strength, on the one hand, of the abnormal stimulus to heat production arising from the devitalisation of the inflamed part, and, on the other hand, of the force which the nervous system of the individual can employ for the purpose of maintaining the normal thermal balance.

This view is supported by the fact that the nearer an inflammation goes to causing sloughing, the higher does the temperature rise; whereas, when a line of demarcation takes place, the temperature falls. Thus it is not the "death," but the "dying" of the part which raises the temperature. It is to be noted that, according to this view, the condition which causes the rise of temperature is not a simple stimulation of nerves to increased function, but an altogether abnormal irritation of the nerves of the part.

If this be the correct explanation of the elevation of temperature in traumatic fever, it seems not improbable that the nervous tract to the inner side of the corpus striatum, the stimulation of which gives rise to an elevation of temperature, is not an efferent, but an afferent tract, or that it at least implicates afferent fibres, the stimulation of which conveys to some heat-controlling nervous centre a message of coldness which does not exist. The stimulation of such fibres would produce an action in opposition to that which normally prevents the body heat from rising above its healthy

standard. Such stimulation would, therefore, tend to produce a rise of temperature. Stimulation of the heat centre itself would necessarily involve both afferent and efferent fibres.

In the foregoing pages, although the alterations of the pulse and of the temperature in fever have been attributed to the action of separate physiological mechanisms, they are traced to exactly the same cause—the partial devitalisation of the inflamed part. Hence all the conditions which tend to increase or diminish local inflammatory action will influence simultaneously the pulse and the temperature. If, in an individual case, the pulse changes are more prominent than the temperature changes, or *vice versâ*, the explanation is to be found in a greater or less strength of the cardiac muscle, in a greater or less stability of the vaso-motor or heat-regulating nervous centres, or in certain other special conditions. It is admitted, however, that in inflammation the changes in pulse and temperature do tend to take place together,—to diverge from the normal, and to return to the same synchronously; and it has been brought out sufficiently clearly by clinical evidence that if this does not occur there is often, if not always, some appreciable physiological or pathological peculiarity, or some complication, to account for the exception.

Conclusions as regards the Effects of a Wound on the Vascular and Thermal Systems.

The foregoing views of the effects of a wound may be shortly summed up as follows. An operation invariably gives rise to a partial devitalisation of the divided tissues. In the vessels of these tissues there is a certain amount of stasis or hindrance to the flow of blood. There is also an exudation of white blood-corpuscles and serum into the injured and surrounding parts, while, in the devitalised centre, and to a varying extent beyond it, there is a dilatation of the blood-vessels. Until the condition of devitalisation begins to be recovered from, there is an extension of the area of stasis and exudation. The devitalised condition tends to pass off in from two to three days. Then the hindrance to the blood-flow ceases, the exudations are reabsorbed, the vessels recover their proper calibre, and everything returns to the normal state.

The condition of the irritated tissues affects both the vascular and the thermal systems. As regards the vascular system, the pathological interference with the supply of blood to the injured parts causes a reflex physiological contraction of the arteries throughout the rest of the body, and consequently a determination of blood to the inflamed area, which is permitted by the local dilatation of

the vessels surrounding the damaged parts. As the arteries throughout the body contract, the blood-supply to the tissues generally is diminished, and an universal reflex stimulus to vascular dilatation is thus produced. This tends to control the amount of vascular contraction.

The diminution of the vascular capacity brought about by the contraction of the arteries necessitates a reduction of the quantity of blood in them. Consequently, in the absence of venous congestion, the tension of the fluids in the lymphatics and tissues generally must be raised. In healthy conditions this tension is relieved by a profuse action of the sweat glands, and the more fluid portions of the blood are got rid of. At the same time an excessive destruction of red blood-corpuscles takes place, and their constituent parts are eliminated in the bile and urine. Much of the blood is thus broken up and excreted, so that the total quantity of this fluid is diminished. The loss brought about in this way gives rise reflexly to an increased demand for fresh blood, and consequent formation of it, which drains the stores of pabulum laid up in the various tissues, and leads to a rapid absorption of fat, great wasting of the muscles, and corresponding weakness. So long as the blood-vessels remain contracted, excessive destruction and compensatory formation of blood continue. The unusual amount of chemical reaction thus in-

duced accounts for the increase of the production and elimination of heat and waste products, which is found throughout a febrile attack, but does not account for the rise of temperature in fever.

The devitalisation of the tissues also induces a local physiological inactivity, and would cause a local coldness but for the action of the more healthy parts. This, which is a pathological condition, stimulates heat production through the normal physiological, heat-regulating mechanism, and the stimulation becomes stronger as long as the area of devitalisation caused by the irritation increases, and also in proportion to the near approach to actual death of the injured parts. There is induced a local demand for more heat, and the vascular conditions are well calculated to bring any extra heat that is produced as much as possible into the inflamed area. But whenever the tissues are raised above their usual temperature, those nerves which announce to the thermal centre the amount of thermolysis necessary to maintain the normal body heat are stimulated; and according to the power with which thermolysis is induced the temperature is controlled. In proportion as the irritated parts regain their vitality the abnormal stimulation of heat production diminishes, and the full power of the nervous centres which regulate the body temperature in health is re-established.

These changes occur when a healthy inflammation takes place in a healthy patient and undergoes resolution in a healthy way. If, at any time in the course of such an inflammation, some condition should arise which induces a further contraction of the arteries, and if the complication be sufficiently continuous and severe, death is brought about by the shutting off of the blood-supply from the various organs, and finally from the medulla oblongata. As this process takes place, the blood-supply to the inflamed parts also becomes diminished. Passive congestion in the healing tissues occurs, and resolution is interfered with. Inflammatory action tends to return, to spread, and reflexly to produce a rise of temperature, and to increase to a still greater extent the contraction of the blood-vessels. The heart beats to the last, and may even struggle to carry on its work after the medulla has ceased to act.

The foregoing conclusions apply to the simplest form of fever only. In simple cases it is possible to perform operations of great magnitude and severity with very little rise of temperature as a consequence. So much so that many surgeons claim that a considerable proportion of their operation cases recover without any fever at all. They can quote authority that a temperature below 100·4° F., in the axilla, proves the absence

of fever at the moment of observation.* If this authority be trustworthy, there are indeed many operations that are recovered from without fever. But as the pathology of this process has become better understood, it has been recognised that "high temperature is not necessarily fever, and fever is not necessarily accompanied by high temperature. . . . . It is the excessive thermogenesis, with the excessive catabolism of nitrogenous tissue which that involves, that constitutes fever."† Thus, though, after a major operation, the temperature may remain low, evidences of the changes above described as occurring in the vascular system may usually be found. Excretion from the kidneys is markedly increased, and sweating is profuse. When a rapid removal of waste products takes place, the tension in the tissues and vessels being immediately relieved, there remains no condition tending to aggravate the effects of the wound, and therefore the temperature and pulse show little alteration.

The Mode of Action of the Poisons of Specific and Septic Fevers.

In every form of fever all the conditions which have been described above may be detected. In

* Wunderlich's 'Medical Thermometry,' p. 205.

† Macalister, 'Brit. Med. Journ.,' 1887, vol. i, p. 670.

the specific fevers and in the septic fevers there is evidence that some process is taking place besides and beyond the conditions which constitute a simple fever arising from an injury. The superadded causal condition is a poison in the system—a specific poison giving rise to the pathognomonic signs of the particular disease which is produced. Of the fevers thus brought about, the septic varieties are specially deserving the attention of the surgeon.

The septic fevers give rise to the condition called septicæmia. This term should only be applied to a complication of traumatic fever due to the absorption of a poison through a wound, or to the development of a poison in a wound, and its subsequent absorption. The admission of a poison to the tissues through a mucous membrane, as described by Olshausen and Verchère in cases of pseudo-ileus following a laparotomy, has been regarded by some as a mode of development of septicæmia. In the foregoing pages reasons have been given for believing that in the case instanced the symptoms and death are due to other causes. Cases, however, may and do occur in which a poison gains entrance into the system during a traumatic inflammatory attack, but in which the condition produced is not, at least need not be, caused by the absorption of a poison through a wound, or in any way due to

the presence of a wound. It is supposed by some that a patient who has recently undergone an operation, and a lying-in woman, are more susceptible to the exanthemata than other people are. Notwithstanding this, if a patient during convalescence from an operation should be seized by a specific fever other than surgical septicæmia, the condition arising ought, if possible, to be clearly separated from those conditions in which a poison that could not otherwise be harmful is developed in, or absorbed from a wound. Cases having such an essential difference as regards their ætiology should not be classed together under one name, however great the clinical resemblances between them may be.

Even when restricted to conditions arising directly from the absorption of a poison through a wound, the term septicæmia comprises several pathological states. There are at least three well-differentiated forms of this disease. It is known that a substance may be extracted from putrefying animal matter, which, when introduced into the blood-stream, causes death with the ordinary symptoms of septicæmia, but without any evidence of the further development of a poison in the blood or tissues, so that a drop of the blood of an animal killed by this substance is not poisonous when introduced into the vascular system of a healthy animal. The disease induced

in this case is called septic intoxication, or sapræmia.

In a second form, to which the term septicæmia is more properly restricted, microscopic living organisms are found in the blood and in all the tissues of the body. The most minute portion of the blood of an animal dying from this poison, if injected into the tissues of a healthy animal, will communicate the disease to the latter, the blood and tissues of which are found after death to be also permeated by living organisms. This proves that these organisms multiply in the tissues of their victim.

In a third form—pyæmia—the patient suffers from abscesses in many parts of the body, pus formation taking place again and again. Death is brought about by acute febrile disturbance, or by sheer exhaustion, but recovery sometimes takes place after a more or less prolonged illness. This form is also associated with the development of micro-organisms in the tissues, and is extremely infectious.

When death is produced by septicæmia within three or four days of an operation, the clinical symptoms of these three forms of the disease may not be separable except by chemical, microscopic, culture, and experimental examination of the secretions, discharges, and blood.

The symptoms of a fatal surgical septicæmia

are those of an universal irritation added to those of a traumatic fever. There is delirium with excitement, while muscular cramps and twitchings are common. Albuminuria is a characteristic condition, sometimes with evidences of acute nephritis, and in fatal cases suppression of urine is almost constant. Vomiting and diarrhœa, which may be bloody, are frequently observed. The temperature is usually high, but may fall almost as in shock. The pulse becomes gradually smaller and faster, and emaciation is extreme.

The post-mortem appearances also indicate universal irritation. Except in cases of septic intoxication, micro-organisms have been found in all the tissues of the body; but of all the organs, the kidneys are most constantly affected by them. Clinical evidence supports the view that the elimination of the poison is mainly effected through the renal tissue ; for in all forms of the disease, if the kidneys continue to act, there is a possibility, even a probability, that the patient may recover. But if albuminuria and suppression of urine occur, and are at all continuous, death will speedily follow. We cannot, however, attribute death from septicæmia, with suppression of urine after an operation, to retention of physiological effete matter which ought to be excreted. As was pointed out in regard to obstruction of the bowels

after an operation, so in this case, death is too rapid to be due to the complication alone. Septicæmic suppression of urine rarely lasts more than three days without being fatal, whereas life is usually prolonged in simple obstructive suppression of urine, even when complete, to from nine to eleven days.* It is obvious on post-mortem examination that the suppression of the secretion of urine in these cases is associated with passive congestion of the kidneys, and there is evidence both in the urine and in the post-mortem conditions that there is much irritation of the kidney substance, sometimes resulting in acute nephritis. These conditions must cause a great obstruction to the flow of blood through the kidneys. When this occurs, there arises a stimulus to physiological action in every respect parallel to that which exists when the blood-flow through the mesenteric vessels is interfered with by intestinal distension in cases of obstruction or paralysis of the bowel after a laparotomy. Just as in a pseudo-ileus arising during a healing process, so in suppression of urine from irritation of the kidneys, occurring in the course of a traumatic fever, the opposition to the flow of blood will induce reflexly an increased contraction of the systemic vessels. If the cause be sufficiently powerful, the death of the patient must be brought

* Roberts, 'Urinary and Renal Diseases,' p. 27.

about by irritation of the kidneys in exactly the same way as has been described as occurring in pseudo-ileus. There are, therefore, many resemblances between the two classes of cases. Nevertheless the only cause of the exacerbation of the inflammatory fever which is common to pseudo-ileus after laparotomy and to suppression of urine in septicæmia, is the obstruction to the flow of blood through a large and important vascular area.

Sufficient grounds for a differential diagnosis between cases of septicæmia and cases of pseudo-ileus may usually be found, and the differences in the symptoms are fully explained by their causes. So like, however, are the symptoms of these diseases in many respects that until recently they have been altogether confounded with one another in abdominal surgery. Even now Olshausen and Verchère, though recognising that there are some differences between these two pathological states, have attributed the symptoms of pseudo-ileus to a species of septicæmia peculiar to that condition.

If from any cause the skin does not act during a traumatic fever, the blood-pressure may be raised after the same fashion as in pseudo-ileus and irritative suppression of urine, and may so lead to a great increase of the febrile symptoms, or even to death. A severe inflammation, such

as an intense pneumonia following on an operation, must tend to produce a similar effect. Such a complication has, however, a natural tendency to recover, and therefore is not necessarily fatal.

Certain drugs, notably turpentine, copaiba, and opium, and many alkaloids, may also produce irritative suppression of urine and even acute nephritis. It is probable that an interference with the renal secretion brought about by the action of one of these drugs or alkaloids, after an operation, if lasting sufficiently long, would prove fatal, with symptoms resembling in many respects those of a true septicæmia and of a pseudo-ileus. So closely may septicæmia be simulated by a variety of conditions that the differential diagnosis is in many cases difficult, and may even be impossible with our present knowledge.

It may, however, be stated generally that any complication of traumatic fever which raises the blood-pressure, or interferes with the elimination of waste products, and so indirectly raises the blood-pressure, is dangerous, and may induce death in the manner which has been described. Some at least of the complications which act in this way are not inflammatory, and are therefore not accompanied by a rise of temperature until fresh inflammatory action is induced in the wound. In this fact we have an explanation of

the serious importance which all surgeons of experience attach to the combination of a rising pulse with a falling temperature. This combination of symptoms often indicates that inflammation is not the chief cause of mischief, but that there is a complication somewhere (often in the kidneys, and in abdominal surgery frequently an ileus or a pseudo-ileus) which is causing an increased contraction of the arteries, and which, if persistent, will induce a fatal aggravation of the inflammatory action.

By the interpretation given in the preceding pages of the symptoms in pseudo-ileus, in septicæmia, and in other complications of traumatic fever we can also fully account for some of those anomalous cases in which it has appeared as if septicæmia (traumatic blood-poisoning) could be induced by contact with the exanthemata. The explanation is to be found in the fact that many of the contagious fevers—diphtheria and scarlet fever, for instance—are prone to be complicated by irritative suppression of urine. If it be remembered that after an operation the kidneys have a great deal of extra work thrown upon them, it will be intelligible that any disease, the poison of which is eliminated by the kidneys, or is liable to irritate the kidneys, will be much more likely to be attended with suppression of urine if complicated by a traumatic fever than if

it occurred in an otherwise healthy subject. Hence, suppression of urine may be brought about by a specific poison, and the patient may be killed by exacerbation of the inflammatory condition before any pathognomonic symptoms of the specific disease arising from the poison are detectable, but with sufficient evidence in the condition of the mind and of the body that some poison is at work. Under these circumstances it would be impossible to distinguish clinically the symptoms produced from those which would be caused by the action on the kidneys of a poison absorbed through the wound in a case of true surgical septicæmia. These considerations, moreover, suggest the possibility that in the increased work thrown on the kidneys during a traumatic fever may be found the explanation of the fact, if it be a fact, that operation cases and women during convalescence from childbirth are specially liable to be attacked by the exanthemata. It may be that a dose of the poison which in health would be excreted without any symptoms is arrested by the overworked kidneys of a febrile subject, and so gives rise to its specific disease.

Those who do not attribute the changes of temperature occurring in even the simplest fever to peripheral irritation, assert that the phenomena observed are always due to the action of some poisonous substance circulating in the system.

Without doubt poisons so circulating do cause fever. The question is whether they cause it by a specific action on some nerve-centre, or by a wide-spread effect on the tissues generally. It seems highly probable that a series of phenomena such as that of fever, tending to show itself in a large variety of diseases with almost constant uniformity, must have some cause common to all cases; while the many symptoms which occur in some only of the various fevers, and those other symptoms which are at times present and at times absent, are obviously not due to essential elements in the febrile process.

If it be concluded that peripheral irritation is not the cause of fever, both the hypothetical substance which produces this disturbance and its mode of action have yet to be discovered. On the other hand, if the views of traumatic fever which the writer has endeavoured to formulate are correct, they also explain how certain irritant poisons circulating in the system may cause all the characteristic phenomena of the febrile state. Many of the constituents of the discharges from a recent wound—*e. g.* partly clotted blood, simple serum, serous exudation, blood-pigments (hæmoglobin, &c.), and blood-ferment (Schmidt),—are pyrogenous when injected into the blood-stream of a healthy animal.*

* Victor Horsley, 'Heath's Dictionary of Surgery,' p. 368.

Other substances, such as pus and unhealthy discharges, and many other ferments, act in the same way. These substances are of low vital power, or without life, or they may possess a life which is inimical to that of the tissues of their host. One would expect them, when circulating in the system, to give rise to a wide-spread irritation and consequent partial devitalisation of tissue. Such a wide-spread partial devitalisation of tissue must produce the same effects on the vascular and thermal systems as are brought about by the more obvious devitalisation due to a local irritation, such as the infliction of a wound or the application of a caustic. The effect may be slight, or so severe as rapidly to induce great contraction of the arteries, a high temperature, and death. It is even conceivable that death may be produced almost with the suddenness of shock. In accordance with this view we know that, in scarlet fever for example, a fatal result is sometimes brought about by the poison before any pathognomonic signs of the disease show themselves, while the cold stage of cholera may develop and prove fatal within a very short time. Certain snake poisons also kill with extreme rapidity. The peculiarities of the irritant, and the duration of its action, with the special susceptibilities of individuals and of the different parts of the body, fully account for the con-

ditions found in the special diseases produced. In many febrile diseases there are evidences of peripheral mischief. Sapræmia, septicæmia, pyæmia, and all the exanthemata show signs of a wide-spread irritation with a tendency to inflammation in various tissues and organs. In others, such as ague, there is no lesion discovered. Assuming, however, for a moment that the views of the causation of traumatic fever now brought forward are correct, an attack closely resembling that of an ague might be expected to follow an intense general irritation of the tissues of short duration, and not sufficiently severe to cause death of the affected parts, or death of the whole body from shock. Such an irritation would leave no trace of itself, whether it was recovered from or whether it proved fatal. Very similar symptoms—rigor, high temperature, profuse sweating, and complete recovery in a few hours—may sometimes be observed after the passage of a gall-stone through the common bile-duct.

It seems probable that even the presence in the vessels of blood which has been temporarily removed from the body, and therefore temporarily exposed to devitalising influences, is sufficient to induce fever, for transfusion of blood is followed by "a greater or less *febrile reaction*, according to the amount of blood transfused."* In this

* Landois and Stirling, 4th edit., vol. i, p. 164.

case the explanation may be that the partially devitalised portions of the blood produce all the reflex nervous changes which have been attributed in these pages to the partial devitalisation of wounded tissues.

PRINTED BY ADLARD AND SON,
BARTHOLOMEW CLOSE, E.C., AND 20, HANOVER SQUARE, W.

A SELECTION

FROM

J. & A. CHURCHILL'S CATALOGUE,

COMPRISING

MOST OF THE RECENT WORKS PUBLISHED BY THEM.

N.B.—J. & A. Churchill's larger Catalogue, which contains over 600 works, with a Complete Index to their Subjects, will be sent on application.

Human Anatomy:
A Treatise by various Authors. Edited by HENRY MORRIS, M.A., M.B. Lond., F.R.C.S. Roy. 8vo, with 791 Illustrations, nearly all original, and many of them in several colours, 40s.

Heath's Practical Anatomy:
A Manual of Dissections. Eighth Edition. Edited by WILLIAM ANDERSON, F.R.C.S., Surgeon and Lecturer on Anatomy at St. Thomas's Hospital, Examiner in Anatomy for R.C.P. and S. Crown 8vo, with 329 Engravings, 15s.

Wilson's Anatomist's Vade-Mecum. Eleventh Edition. By HENRY E. CLARK, M.R.C.S. Eng., F.F.P.S. Glasg., Professor of Anatomy in St. Mungo's College, Glasgow. Crown 8vo, with 492 Engravings and 26 Coloured Plates, 18s.

An Atlas of Human Anatomy.
By RICKMAN J. GODLEE, M.S., F.R.C.S., Surgeon and late Demonstrator of Anatomy, University College Hospital. With 48 Imp. 4to Plates (112 figures), and a volume of Explanatory Text. 8vo, £4 14s. 6d.

Human Osteology.
By LUTHER HOLDEN, Consulting Surgeon to St. Bartholomew's Hospital. Seventh Edition, edited by CHARLES STEWART, Conservator of the Museum R.C.S., and ROBERT W. REID, M.D., F.R.C.S., Professor of Anatomy in the University of Aberdeen. 8vo, with 59 Lithographic Plates and 75 Engravings. 16s.

Also.

Landmarks, Medical and Surgical. Fourth Edition. 8vo, 3s. 6d.

The Student's Guide to Surgical Anatomy. By EDWARD BELLAMY, F.R.C.S. and Member of the Board of Examiners. Third Edition. Fcap. 8vo, with 81 Engravings. 7s. 6d.

Diagrams of the Nerves of the Human Body, exhibiting their Origin, Divisions, and Connections, with their Distribution to the Various Regions of the Cutaneous Surface, and to all the Muscles. By Sir W. H. FLOWER, K.C.B., F.R.S., F.R.C.S. Third Edition, with 6 Plates. Royal 4to, 12s.

Pathological Anatomy of Diseases. Arranged according to the nomenclature of the R.C.P. Lond. (Student's Guide Series). By NORMAN MOORE, M.D., F.R.C.P., Assistant Physician and Lecturer on Pathological Anatomy to St. Bartholomew's Hospital. Fcap. 8vo, with 111 Engravings, 8s. 6d.

A Manual of Clinical and Practical Pathology. By W. E. WYNTER, M.D., M.R.C.P., F.R.C.S., Medical Registrar to Middlesex Hospital, and F. J. WETHERED, M.D., M.R.C.P., Assistant Physician to Victoria Park Hospital. With 4 Coloured Plates and 67 Engravings. 8vo, 12s. 6d.

Lectures on Pathology:
Delivered at the London Hospital. By the late HENRY GAWEN SUTTON, M.B., F.R.C.P., Physician to, and Lecturer on Pathology at, the London Hospital. Edited by MAURICE E. PAUL, M.D., and Revised by SAMUEL WILKS, M.D., LL.D., F.R.S. 8vo, 15s.

General Pathology:
An Introduction to. By JOHN BLAND SUTTON, F.R.C.S., Sir E. Wilson Lecturer on Pathology, R.C.S.; Assistant Surgeon to, and Lecturer on Anatomy at, Middlesex Hospital. 8vo, with 149 Engravings, 14s.

Atlas of Pathological Anatomy. By Dr. LANCEREAUX. Translated by W. S. GREENFIELD, M.D., Professor of Pathology in the University of Edinburgh. Imp. 8vo, with 70 Coloured Plates, £5 5s.

Atlas of the Central Nervous System. From the larger work of Hirschfeld and Léveillé. Edited by HOWARD H. TOOTH, M.D., F.R.C.P., Assistant Physician to the National Hospital for the Paralysed and Epileptic. With 37 Plates carefully coloured by Hand. Large Imp. 8vo, 40s.

The Human Brain:
Histological and Coarse Methods of Research. A Manual for Students and Asylum Medical Officers. By W. BEVAN LEWIS, L.R.C.P. Lond., Medical Superintendent, West Riding Lunatic Asylum. 8vo, with Wood Engravings and Photographs, 8s.

Elements of Human Physiology. (Student's Guide Series.) By ERNEST H. STARLING, M.D., M.R.C.P., Joint Lecturer on Physiology at Guy's Hospital. Fcap. 8vo, with 94 Engravings, 6s. 6d.

Manual of Physiology:
For the use of Junior Students of Medicine. By GERALD F. YEO, M.D., F.R.S., Emeritus Professor of Physiology in King's College, London. Third Edition. Crown 8vo, with 254 Engravings (many figures), and Plate of Spectra, 14s.

Principles of Human Physiology. By W. B. CARPENTER, C.B., M.D., F.R.S. Ninth Edition. By HENRY POWER, M.B., F.R.C.S. 8vo, with 3 Steel Plates and 377 Wood Engravings, 31s. 6d.

Medical Jurisprudence:
Its Principles and Practice. By ALFRED S. TAYLOR, M.D., F.R.C.P., F.R.S. Third Edition, by THOMAS STEVENSON, M.D., F.R.C.P., Lecturer on Medical Jurisprudence at Guy's Hospital. 2 vols. 8vo, with 188 Engravings, 31s. 6d.

By the same Authors.

A Manual of Medical Jurisprudence. Twelfth Edition. Crown 8vo, with 55 Engravings, 14s.

The Student's Guide to Medical Jurisprudence. By JOHN ABERCROMBIE, M.D., F.R.C.P., Physician to Charing Cross Hospital. Fcap. 8vo, 7s. 6d.

Hospitals, Infirmaries, and Dispensaries: Their Construction, Interior Arrangement, and Management; with Descriptions of existing Institutions, and 74 Illustrations. By F. OPPERT, M.D., M.R.C.P.L. Second Edition. Royal 8vo, 12s.

Sanitary Examinations
Of Water, Air, and Food. A Vade-Mecum for the Medical Officer of Health. By CORNELIUS B. FOX, M.D., F.R.C.P. Second Edition. Crown 8vo, with 110 Engravings, 12s. 6d.

Microscopical Examination of Drinking Water and of Air. By J. D. MACDONALD, M.D., F.R.S., Ex-Professor of Naval Hygiene in the Army Medical School. Second Edition. 8vo, with 25 Plates, 7s. 6d.

Hygiene and Public Health.
A Treatise by various Authors. Edited by THOMAS STEVENSON, M.D., F.R.C.P., Lecturer on Chemistry and Medical Jurisprudence at Guy's Hospital; Official Analyst to the Home Office; and SHIRLEY F. MURPHY, Medical Officer of Health of the County of London. In 2 vols., royal 8vo, fully Illustrated. Vol. I., 28s. Vol. II. *nearly ready.*

A Manual of Practical Hygiene. By the late E. A. PARKES, M.D., F.R.S. Eighth Edition, by J. LANE NOTTER, A.M., M.D., F.R.S., Professor of Military Hygiene in the Army Medical School. 8vo, with 10 Plates and 103 Engravings, 18s.

A Handbook of Hygiene and Sanitary Science. By GEO. WILSON, M.A., M.D., F.R.S.E., D.P.H. Camb., Medical Officer of Health for Mid-Warwickshire. Seventh Edition. Crown 8vo, with Engravings. 12s. 6d.

Public Health Reports.
By Sir JOHN SIMON, C.B., F.R.S. Edited by EDWARD SEATON, M.D., F.R.C.P. 2 vols. 8vo, with Portrait, 36s.

Hospitals and Asylums of the World; their Origin, History, Construction, Administration, Management, and Legislation. By HENRY C. BURDETT. In 4 vols. Super Royal 8vo and Portfolio. Complete, 168s. Vols. I. and II.—Asylums: their History, Administration, and Construction, with Plans and Bibliography, 90s. Vols. III. and IV.—Hospitals, Convalescent Institutions, Nurses' Homes, Medical School Buildings, &c., with Portfolio of Plans, 120s.

Mental Diseases:
Clinical Lectures. By T. S. CLOUSTON, M.D., F.R.C.P. Edin., Lecturer on Mental Diseases in the University of Edinburgh. Third Edition. Crown 8vo, with 13 Plates, 14s.

Illustrations of the Influence of the Mind upon the Body in Health and Disease: Designed to elucidate the Action of the Imagination. By D. HACK TUKE, M.D., F.R.C.P., LL.D. Second Edition. 2 vols. crown 8vo, 15s.

By the same Author.

Prichard and Symonds in Especial Relation to Mental Science. With Chapters on Moral Insanity. 8vo, with 2 Portraits, 5s.

Also.

Reform in the Treatment of the Insane. Early History of the Retreat, York; its Objects and Influence. With a Report of the Celebrations of its Centenary. 8vo, 4s.

A Dictionary of Psychological Medicine, giving the Definition, Etymology, and Synonyms of the Terms used in Medical Psychology; with the Symptoms, Treatment, and Pathology of Insanity; and THE LAW OF LUNACY IN GREAT BRITAIN AND IRELAND. Edited by D. HACK TUKE, M.D., LL.D., assisted by nearly 130 Contributors, British, Continental and American. 2 vols., 1,500 pages, royal 8vo, Illustrated. 42s.

The Journal of Mental Science. Published Quarterly, by Authority of the Medico-Psychological Association. 8vo, 3s. 6d.

Mental Affections of Childhood and Youth (Lettsomian Lectures for 1887, &c.). By J. LANGDON DOWN, M.D., F.R.C.P., Consulting Physician to the London Hospital. 8vo, 6s.

Manual of Midwifery.
By ALFRED L. GALABIN, M.A., M.D., F.R.C.P., Obstetric Physician to, and Lecturer on Midwifery, &c., at, Guy's Hospital. Second Edition. Crown 8vo, with 249 Engravings, 15s.

The Student's Guide to the Practice of Midwifery. By D. LLOYD ROBERTS, M.D., F.R.C.P., Lecturer on Clinical Midwifery and Diseases of Women at the Owens College; Obstetric Physician to the Manchester Royal Infirmary. Fourth Edition. Fcap. 8vo, with Coloured Plates and Engravings.
[*In the Press.*

Manual of the Diseases peculiar to Women. By JAMES OLIVER, M.D., F.R.S. Edin., M.R.C.P. Lond., Physician to the Hospital for Women, London. Fcap. 8vo, 3s. 6d.

Obstetric Aphorisms:
For the Use of Students commencing Midwifery Practice. By JOSEPH G. SWAYNE, M.D. Tenth Edition. Fcap. 8vo, with 20 Engravings, 3s. 6d.

Lectures on Obstetric Operations: Including the Treatment of Hæmorrhage, and forming a Guide to the Management of Difficult Labour. By ROBERT BARNES, M.D., F.R.C.P., Consulting Obstetric Physician to St. George's Hospital. Fourth Edition. 8vo, with 121 Engravings, 12s. 6d.

By the same Author.

A Clinical History of Medical and Surgical Diseases of Women. Second Edition. 8vo, with 181 Engravings, 28s.

Clinical Lectures on Diseases of Women: Delivered in St. Bartholomew's Hospital, by J. MATTHEWS DUNCAN, M.D., LL.D., F.R.C.P., F.R.Ss. L. & E., late Obstetric Physician to St. Bartholomew's Hospital. Fourth Edition. 8vo, 16s.

Gynæcological Operations:
(Handbook of). By ALBAN H. G. DORAN, F.R.C.S., Surgeon to the Samaritan Hospital. 8vo, with 167 Engravings, 15s.

The Student's Guide to the Diseases of Women. By ALFRED L. GALABIN, M.A., M.D., F.R.C.P., Obstetric Physician to Guy's Hospital. Fifth Edition. Fcap. 8vo, with 142 Engravings, 8s. 6d.

A Practical Treatise on the Diseases of Women. By T. GAILLARD THOMAS, M.D. Sixth Edition, by PAUL F. MUNDÉ, M.D., Professor of Gynæcology at the New York Polyclinic and at Dartmouth College. Roy. 8vo, with 347 Engravings, 25s.

Abdominal Surgery.
By J. GREIG SMITH, M.A., F.R.S.E., Surgeon to the Bristol Royal Infirmary, and Lecturer on Surgery in the Bristol Medical School. Fourth Edition. 8vo, with 82 Engravings, 21s.

Notes on Gynæcological Nursing. By JOHN BENJAMIN HILLIER, M.D., M.R.C.S., Lecturer on the Diseases of Women and Children in the Yorkshire College, and Surgeon to the Hospital for Women, &c., Leeds. Crown 8vo, 1s. 6d.

A Manual for Hospital Nurses and others engaged in Attending on the Sick, with a Glossary. By EDWARD J. DOMVILLE, Surgeon to the Exeter Lying-in Charity. Seventh Edition. Crown 8vo, 2s. 6d.

A Manual of Nursing, Medical and Surgical. By CHARLES J. CULLINGWORTH, M.D., F.R.C.P., Obstetric Physician to St. Thomas's Hospital. Third Edition. Fcap. 8vo, with Engravings, 2s. 6d.

By the same Author.

A Short Manual for Monthly Nurses. Third Edition. Fcap. 8vo, 1s. 6d.

Diseases of Children.
For Practitioners and Students. By W. H. DAY, M.D., Physician to the Samaritan Hospital. Second Edition. Crown 8vo, 12s. 6d.

The Diseases of Children (Student's Guide Series). By JAS. F. GOODHART, M.D., F.R.C.P., Physician to Guy's Hospital. Fourth Edition. Fcap. 8vo, 10s. 6d.

A Practical Treatise on Disease in Children. By EUSTACE SMITH, M.D., F.R.C.P., Physician to the King of the Belgians, and to the East London Hospital for Children, &c. Second Edition. 8vo, 22s.

By the same Author.

Clinical Studies of Disease in Children. Second Edition. Post 8vo, 7s. 6d.

Also.

The Wasting Diseases of Infants and Children. Fifth Edition. Post 8vo, 8s. 6d.

A Practical Manual of the Diseases of Children. With a Formulary. By EDWARD ELLIS, M.D. Fifth Edition. Crown 8vo, 10s.

Materia Medica:
A Manual for the use of Students. By ISAMBARD OWEN, M.D., F.R.C.P., Lecturer on Materia Medica, &c., to St. George's Hospital. Second Edition. Crown 8vo, 6s. 6d.

Materia Medica,
Pharmacy, Pharmacology, and Therapeutics. By W. HALE WHITE, M.D., F.R.C.P., Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; Examiner in Materia Medica on the Conjoint Board of the Royal Colleges of Physicians and Surgeons. Fcap. 8vo, 7s. 6d.

Materia Medica
And Therapeutics. By CHARLES D. F. PHILLIPS, M.D., F.R.S. Edin. Vegetable Kingdom—Organic Compounds—Animal Kingdom. 8vo, 25s.

Organic Materia Medica
Of the British Pharmacopœia, systematically arranged; with Notices of Remedies in the Indian and U.S. Pharmacopœias, and Descriptions of Adulterants and Substitutions. By W. SOUTHALL, F.L.S. Fourth Edition. Crown 8vo, 5s.

Recent Materia Medica.
Notes on their Origin and Therapeutics. By F. HARWOOD LESCHER, F.C.S., Pereira Medallist. Fourth Edition. 8vo, 2s. 6d

Galenic Pharmacy:
A Practical Handbook to the Processes of the British Pharmacopœia. By R. A. CRIPPS, M.P.S. 8vo, with 76 Engravings, 8s. 6d.

Practical Pharmacy.
By BARNARD S. PROCTOR, formerly Lecturer on Pharmacy at the College of Medicine, Newcastle-on-Tyne. Third Edition. 8vo, with 44 Wood Engravings and 32 Lithograph Fac-Simile Prescriptions, 14s.

Selecta è Prescriptis:
Containing Terms, Phrases, Contractions and Abbreviations used in Prescriptions with Explanatory Notes, &c. Also, a Series of Abbreviated Prescriptions with Translations and Key. By J. PEREIRA, M.D., F.R.S. Eighteenth Edition, by JOSEPH INCE, F.C.S., F.L.S. 24mo, 5s.

Pocket Formulary
And Synopsis of the British and Foreign Pharmacopœias. By HENRY BEASLEY. Eleventh Edition. 18mo, 6s. 6d.

By the same Author.

Druggist's General Receipt Book. Ninth Edition. 18mo, 6s. 6d.

Also.

Book of Prescriptions:
Containing upwards of 3,000 Prescriptions from the Practice of the most eminent Physicians and Surgeons, English and Foreign. Seventh Edition. 18mo, 6s. 6d.

A Companion to the British Pharmacopœia. By PETER SQUIRE. Revised by his Sons, P. W. and A. H. SQUIRE. Fifteenth Edition. 8vo, 10s. 6d. Supplement, 1s.

By the same Authors.

The Pharmacopœias of the London Hospitals, arranged in Groups for Easy Reference and Comparison. Sixth Edition. 18mo. 6s.

The Prescriber's Pharmacopœia: The Medicines arranged in Classes according to their Action, with their Composition and Doses. By NESTOR J. C. TIRARD, M.D., F.R.C.P., Professor of Materia Medica and Therapeutics in King's College, London. Sixth Edition. 32mo, bound in leather, 3s.

Year-Book of Pharmacy: Containing the Transactions of the British Pharmaceutical Conference. Annually. 8vo, 10s.

Royle's Manual of Materia Medica and Therapeutics. Sixth Edition, including additions and alterations in the B.P. 1885. By JOHN HARLEY, M.D., Physician to St. Thomas's Hospital. Crown 8vo, with 139 Engravings, 15s.

Manual of Botany: Including the Structure, Classification, Properties, Uses, and Functions of Plants. By ROBERT BENTLEY, Emeritus Professor of Botany in King's College and to the Pharmaceutical Society. Fifth Edition. Crown 8vo, with 1,178 Engravings, 15s.

By the same Author.

The Student's Guide to Structural, Morphological, and Physiological Botany. With 660 Engravings. Fcap. 8vo, 7s. 6d.

Also.

The Student's Guide to Systematic Botany, including the Classification of Plants and Descriptive Botany. Fcap. 8vo, with 350 Engravings, 3s. 6d.

Medicinal Plants: Being descriptions, with original figures, of the Principal Plants employed in Medicine, and an account of their Properties and Uses. By Prof. BENTLEY and Dr. H. TRIMEN, F.R.S. In 4 vols., large 8vo, with 306 Coloured Plates, bound in Half Morocco, Gilt Edges, £11 11s.

Elementary Practical Biology. Vegetable. By THOMAS W. SHORE, M.D., B.Sc. Lond., Lecturer on Comparative Anatomy at St. Bartholomew's Hospital. 8vo, 6s.

Climate and Fevers of India, with a series of Cases (Croonian Lectures, 1882). By Sir JOSEPH FAYRER, K.C.S.I., M.D. 8vo, with 17 Temperature Charts, 12s.

By the same Author.

The Natural History and Epidemiology of Cholera; Being the Annual Oration of the Medical Society of London, 1888. 8vo, 3s. 6d.

A Manual of Family Medicine and Hygiene for India. Published under the Authority of the Government of India. By Sir WILLIAM J. MOORE, K.C.I.E., M.D., late Surgeon-General with the Government of Bombay. Sixth Edition. Post 8vo, with Engravings, 12s.

By the same Author.

A Manual of the Diseases of India: With a Compendium of Diseases generally. Second Edition. Post 8vo, 10s.

Also,

The Constitutional Requirements for Tropical Climates, &c. Crown 8vo, 4s.

The Prevention of Disease in Tropical and Sub-Tropical Campaigns. (Parkes Memorial Prize for 1886.) By ANDREW DUNCAN, M.D., B.S. Lond., F.R.C.S., Surgeon-Major, Bengal Army. 8vo, 12s. 6d.

Practical Therapeutics: A Manual. By EDWARD J. WARING, C.I.E., M.D., F.R.C.P., and DUDLEY W. BUXTON, M.D., B.S. Lond. Fourth Edition. Crown 8vo, 14s.

By the same Author.

Bazaar Medicines of India, And Common Medical Plants: With Full Index of Diseases, indicating their Treatment by these and other Agents procurable throughout India, &c. Fourth Edition Fcap. 8vo, 5s.

A Commentary on the Diseases of India. By NORMAN CHEVERS, C.I.E., M.D., F.R.C.S., Deputy Surgeon-General H.M. Indian Army. 8vo, 24s.

Hooper's Physicians' Vade-Mecum. A Manual of the Principles and Practice of Physic. Tenth Edition. By W. A. GUY, F.R.C.P., F.R.S., and J. HARLEY, M.D., F.R.C.P. With 118 Engravings. Fcap. 8vo, 12s. 6d.

The Principles and Practice of Medicine. (Text-book.) By the late C. HILTON FAGGE, M.D., and P. H. PYE-SMITH, M.D., F.R.S., F.R.C.P., Physician to, and Lecturer on Medicine in, Guy's Hospital. Third Edition. 2 vols. 8vo, cloth, 40s.; Half Leather, 46s.

A Dictionary of Practical Medicine. By various writers. Edited by JAS. KINGSTON FOWLER, M.A., M.D., F.R.C.P., Physician to Middlesex Hospital and the Hospital for Consumption. 8vo, cloth, 21s.; half calf, 25s.

Manual of the Practice of Medicine. By FREDERICK TAYLOR, M.D., F.R.C.P., Physician to, and Lecturer on Medicine at, Guy's Hospital. Third Edition. Cr. 8vo, with Engravings, 15s.

The Practice of Medicine (Student's Guide Series). By M. CHARTERIS, M.D., Professor of Therapeutics and Materia Medica in the University of Glasgow. Sixth Edition. Fcap. 8vo, with Engravings on Copper and Wood, 9s.

How to Examine the Chest: A Practical Guide for the use of Students. By SAMUEL WEST, M.D., F.R.C.P., Assistant Physician to St. Bartholomew's Hospital. Second Edition. With Engravings. Fcap. 8vo, 5s.

The Bronchi and Pulmonary Blood-vessels: their Anatomy and Nomenclature. By WILLIAM EWART, M.D., F.R C.P., Physician to St. George's Hospital. 4to, with 20 Illustrations, 21s.

An Atlas of the Pathological Anatomy of the Lungs. By the late WILSON FOX, M.D., F.R.S., F.R.C.P., Physician to H.M. the Queen. With 45 Plates (mostly Coloured) and Engravings. 4to, half-bound in Calf, 70s.

By the same Author.

A Treatise on Diseases of the Lungs and Pleura. Edited by SIDNEY COUPLAND, M.D., F.R.C.P., Physician to Middlesex Hospital. Roy. 8vo, with Engravings; also Portrait and Memoir of the Author, 36s.

The Student's Guide to Diseases of the Chest. By VINCENT D. HARRIS, M.D. Lond., F.R.C.P., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park. Fcap. 8vo, with 55 Illustrations (some Coloured), 7s. 6d.

Antiseptic Dry-Air Treatment of Consumption. By JOHN J. HARTNETT, M.D., M.Ch., Q.U.I. With Engravings. Crown 8vo, 3s. 6d.

Guy's Hospital Reports. By the Medical and Surgical Staff. Vol. XXXIV. Third Series. 8vo, 10s. 6d.

St. Thomas's Hospital Reports. By the Medical and Surgical Staff. Vol. XXI. New Series. 8vo, 8s. 6d.

Westminster Hospital Reports. By the Medical and Surgical Staff. Vol. VIII. 8vo, 6s.

The Climate of Rome and the Roman Malaria. By Professor TOMMASI-CRUDELI. Translated by CHARLES CRAMOND DICK. Crown 8vo, 5s.

Medical Diagnosis (Student's Guide Series). By SAMUEL FENWICK, M.D., F.R.C.P., Physician to the London Hospital. Seventh Edition. Fcap. 8vo, with 117 Engravings, 7s.

By the same Author.

Outlines of Medical Treatment. Third Edition. Crown 8vo, 10s.

Also.

Clinical Lectures on Some Obscure Diseases of the Abdomen. Delivered at the London Hospital. 8vo, with Engravings, 7s. 6d.

Also.

The Saliva as a Test for Functional Diseases of the Liver. Crown 8vo, 2s.

The Microscope in Medicine. By LIONEL S. BEALE, M.B., F.R.S., Physician to King's College Hospital. Fourth Edition. 8vo, with 86 Plates, 21s.

By the same Author.

The Liver. With 24 Plates (85 Figures). 8vo. 5s.

Also.

On Slight Ailments: And on Treating Disease. Third Edition. 8vo, 5s.

Medical Lectures and Essays. By Sir G. JOHNSON, M.D., F.R.C.P., F.R.S., Consulting Physician to King's College Hospital. 8vo, with 46 Engravings, 25s.

By the same Author.

An Essay on Asphyxia (Apnœa). 8vo, 3s.

Uric Acid as a Factor in the Causation of Disease. By ALEXANDER HAIG, M.D., F.R.C.P., Physician to the Metropolitan Hospital and the Royal Hospital for Children and Women. With 23 Illustrations, 8vo, 8s. 6d.

Bronchial Affections: Pneumonia and Fibroid Pneumonia (their Pathological Histology). An Original Investigation. By A. G. AULD, M.D., Assistant Physician to the Glasgow Royal Infirmary. 8vo, with Illustrations, 7s. 6d.

Bronchial Asthma: Its Pathology and Treatment. By J. B. BERKART, M.D., late Physician to the City of London Hospital for Diseases of the Chest. Second Edition, with 7 Plates (35 Figures). 8vo, 10s. 6d.

Treatment of Some of the Forms of Valvular Disease of the Heart. By A. E. SANSOM, M.D., F.R.C.P., Physician to the London Hospital. Second Edition. Fcap. 8vo, with 26 Engravings, 4s. 6d.

Vaccinia and Variola:
A Study of their Life History. By JOHN B. BUIST, M.D., F.R.S.E., Teacher of Vaccination for the Local Government Board. Crown 8vo, with 24 Coloured Plates, 7s. 6d.

Medical Ophthalmoscopy:
A Manual and Atlas. By W. R. GOWERS, M.D., F.R.C.P., F.R.S., Physician to the National Hospital for the Paralyzed and Epileptic. Third Edition. Edited with the assistance of MARCUS GUNN, M.B., F.R.C.S., Surgeon to the Royal London Ophthalmic Hospital. With Coloured Plates and Woodcuts. 8vo, 16s.

By the same Author.

A Manual of Diseases of the Nervous System.
Vol. I. Diseases of the Nerves and Spinal Cord. Second Edition. Roy. 8vo, with 179 Engravings, 15s.
Vol. II. Diseases of the Brain and Cranial Nerves: General and Functional Diseases of the Nervous System. Second Edition. Roy. 8vo, with 182 Engravings, 20s.

Also.

Diagnosis of Diseases of the Brain. Second Edition. 8vo, with Engravings, 7s. 6d.

Also.

Syphilis and the Nervous System. Being a Revised Reprint of the Lettsomian Lectures for 1890. Delivered before the Medical Society of London. 8vo, 4s.

The Nervous System,
Diseases of. By J. A. ORMEROD, M.D., F.R.C.P., Physician to the National Hospital for the Paralysed and Epileptic. With 66 Illustrations. Fcap. 8vo, 8s. 6d.

Handbook of the Diseases of the Nervous System. By JAMES ROSS, M.D., F.R.C.P., Professor of Medicine in the Victoria University, and Physician to the Royal Infirmary, Manchester. Roy. 8vo, with 184 Engravings, 18s.

Also.

Aphasia:
Being a Contribution to the Subject of the Dissolution of Speech from Cerebral Disease. 8vo, with Engravings, 4s. 6d.

Aphasia: or Loss of Speech
And the Localization of the Faculty of Articulate Language. By Sir FREDERIC BATEMAN, M.D., F.R.C.P., Senior Physician to the Norfolk and Norwich Hospital. 8vo, 16s.

Diseases of the Nervous System.
Lectures delivered at Guy's Hospital. By SAMUEL WILKS, M.D., F.R.S. Second Edition. 8vo, 18s.

Stammering:
Its Causes, Treatment, and Cure. By A. G. BERNARD, M.R.C.S., L.R.C.P. Crown 8vo, 2s.

Secondary Degenerations of the Spinal Cord (Gulstonian Lectures, 1889). By HOWARD H. TOOTH, M.D., F.R.C.P., Assistant Physician to the National Hospital for the Paralysed and Epileptic. With Plates and Engravings. 8vo, 3s. 6d.

Diseases of the Nervous System.
Clinical Lectures. By THOMAS BUZZARD, M.D., F.R.C.P., Physician to the National Hospital for the Paralysed and Epileptic. With Engravings, 8vo. 15s.

By the same Author.

Some Forms of Paralysis from Peripheral Neuritis: of Gouty, Alcoholic, Diphtheritic, and other origin. Crown 8vo, 5s.

Also.

On the Simulation of Hysteria by Organic Disease of the Nervous System. Crown 8vo, 4s. 6d.

Gout in its Clinical Aspects. By J. MORTIMER GRANVILLE, M.D. Crown 8vo, 6s.

Diseases of the Liver:
With and without Jaundice. By GEORGE HARLEY, M.D., F.R.C.P., F.R.S. 8vo, with 2 Plates and 36 Engravings, 21s.

Rheumatic Diseases,
(Differentiation in). By HUGH LANE, Surgeon to the Royal Mineral Water Hospital, Bath, and Hon. Medical Officer to the Royal United Hospital, Bath. Second Edition, much Enlarged, with 8 Plates. Crown 8vo, 3s. 6d.

Diseases of the Abdomen,
Comprising those of the Stomach and other parts of the Alimentary Canal, Œsophagus, Cæcum, Intestines, and Peritoneum. By S. O. HABERSHON, M.D., F.R.C.P. Fourth Edition. 8vo, with 5 Plates, 21s.

On the Relief of Excessive and Dangerous Tympanites by Puncture of the Abdomen. By JOHN W. OGLE, M.A., M.D., F.R.C.P., Consulting Physician to St. George's Hospital. 8vo, 5s. 6d.

Croonian Lectures on Certain Points connected with Diabetes. By F. W. PAVY, M.D., F.R.S., late Physician to Guy's Hospital. 8vo, 4s. 6d.

Acute Intestinal Strangulation,
And Chronic Intestinal Obstruction (Mode of Death from). By THOMAS BRYANT, F.R.C.S., Senior Surgeon to Guy's Hospital. 8vo, 3s.

Headaches :
Their Nature, Causes, and Treatment. By W. H. DAY, M.D., Physician to the Samaritan Hospital. Fourth Edition. Crown 8vo, with Engravings, 7s. 6d.

Health Resorts at Home and Abroad. By M. CHARTERIS, M.D., Professor of Therapeutics and Materia Medica in Glasgow University. Second Edition. Crown 8vo, with Map, 5s. 6d.

The Mineral Waters of France
And its Wintering Stations (Medical Guide to). With a Special Map. By A. VINTRAS, M.D., Physician to the French Embassy, and to the French Hospital, London. Second Edition. Crown 8vo, 8s.

Canary Islands
Health Resorts, in their Climatological and Medical Aspects. By J. CLEASBY TAYLOR, M.D., M.R.C.S., Las Palmas. 8vo, with Maps, 3s. 6d.

Homburg—Spa.
An Introduction to its Waters and their use. By Dr. ARNOLD SCHETELIG. Crown 8vo, with Synoptical Table, 2s. 6d.

Illustrated Ambulance Lectures :
To which is added a NURSING-LECTURE. By JOHN M. H. MARTIN, M.D., F.R.C.S., Honorary Surgeon to the Blackburn Infirmary. Third Edition. Crown 8vo, with 60 Engravings, 2s.

Surgery : its Theory and Practice (Student's Guide). By WILLIAM J. WALSHAM, F.R.C.S., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital. Fourth Edition. Fcap. 8vo, with 335 Engravings, 12s.

Surgical Emergencies :
Together with the Emergencies attendant on Parturition and the Treatment of Poisoning. By W. PAUL SWAIN, F.R.C.S., Surgeon to the South Devon and East Cornwall Hospital. Fourth Edition. Crown 8vo, with 120 Engravings, 5s.

Operations on the Brain (A Guide to). By ALEC FRASER, Professor of Anatomy, Royal College of Surgeons in Ireland. Illustrated by 42 life-size Plates in Autotype, and 2 Woodcuts in the text. Folio, 63s.

Surgery.
By C. W. MANSELL MOULLIN, M.A., M.D., Oxon., F.R.C.S., Surgeon and Lecturer on Physiology to the London Hospital. Large 8vo, with 497 Engravings, 34s.

A Course of Operative Surgery.
By CHRISTOPHER HEATH, Surgeon to University College Hospital. Second Edition. With 20 coloured Plates (180 figures) from Nature, by M. LÉVEILLÉ, and several Woodcuts. Large 8vo, 30s.

By the same Author.

The Student's Guide to Surgical Diagnosis. Second Edition. Fcap. 8vo, 6s. 6d.

Also.

Manual of Minor Surgery and Bandaging. For the use of House-Surgeons, Dressers, and Junior Practitioners. Ninth Edition. Fcap. 8vo, with 146 Engravings, 6s.

Also.

Injuries and Diseases of the Jaws. Third Edition. 8vo, with Plates and 206 Wood Engravings, 14s.

Also.

Lectures on Certain Diseases of the Jaws. Delivered at the R.C.S., Eng., 1887. 8vo, with 64 Engravings, 2s. 6d.

Also.

Clinical Lectures on Surgical Subjects. Delivered in University College Hospital. Fcap. 8vo, with 23 Engravings, 6s.

The Practice of Surgery :
A Manual. By THOMAS BRYANT, Consulting Surgeon to Guy's Hospital. Fourth Edition. 2 vols. crown 8vo, with 750 Engravings (many being coloured), and including 6 chromo plates, 32s.

By the same Author.

On Tension : Inflammation of Bone, and Head Injuries. Hunterian Lectures, 1888. 8vo, 6s.

The Surgeon's Vade-Mecum :
A Manual of Modern Surgery. By R. DRUITT, F.R.C.S. Twelfth Edition. By STANLEY BOYD, M.B., F.R.C.S. Assistant Surgeon and Pathologist to Charing Cross Hospital. Crown 8vo, with 373 Engravings, 16s.

The Operations of Surgery :
Intended for Use on the Dead and Living Subject alike. By W. H. A. JACOBSON, M.A., M.B., M.Ch. Oxon., F.R.C.S., Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital. Second Edition. 8vo, with 235 Illustrations, 30s.

Diseases of Bones and Joints.
By CHARLES MACNAMARA, F.R.C.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital. 8vo, with Plates and Engravings, 12s.

Lectures on Orthopædic Surgery. By BERNARD E. BRODHURST, F.R.C.S., Surgeon to the Royal Orthopædic Hospital. Second Edition. 8vo, with Engravings, 12s. 6d.

By the same Author.

On Anchylosis, and the Treatment for the Removal of Deformity and the Restoration of Mobility in Various Joints. Fourth Edition. 8vo, with Engravings, 5s.

Also.

Curvatures and Disease of the Spine. Fourth Edition. 8vo, with Engravings, 7s. 6d.

Also.

Nature and Treatment of Talipes Equino-Varus, or Clubfoot. 8vo, with Engravings, 3s. 6d.

Surgical Pathology and Morbid Anatomy (Student's Guide Series). By ANTHONY A. BOWLBY, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital. Second Edition. Fcap. 8vo, with 158 Engravings, 9s.

By the same Author.

Injuries and Diseases of Nerves and their Surgical Treatment. 8vo, with 20 Plates, 14s.

Illustrations of Clinical Surgery. By JONATHAN HUTCHINSON, F.R.S., Senior Surgeon to the London Hospital. In fasciculi. 6s. 6d. each. Fasc. I. to X. bound, with Appendix and Index, £3 10s. Fasc. XI. to XXIII. bound, with Index, £4 10s.

Clubfoot:
Its Causes, Pathology, and Treatment. By WM. ADAMS, F.R.C.S., Consulting Surgeon to the Great Northern and other Hospitals. Second Edition. 8vo, with 106 Engravings and 6 Lithographic Plates, 15s.

By the same Author.

Lateral and other Forms of Curvature of the Spine: Their Pathology and Treatment. Second Edition. 8vo, with 5 Lithographic Plates and 72 Wood Engravings, 10s. 6d.

Also.

Contraction of the Fingers:
(Dupuytren's and Congenital Contractions), their Treatment by Subcutaneous Divisions of the Fascia, and Immediate Extension. Also on **Hammer Toe**; its Curability by Subcutaneous Division. And on **The Obliteration of Depressed Cicatrices** by a Subcutaneous Operation. 8vo, with 8 Plates and 31 Engravings, 6s. 6d.

Treatment of Internal Derangements of the Knee-Joint, by Operation. By HERBERT W. ALLINGHAM, F.R.C.S., Surgeon to the Great Northern Central Hospital, &c. 8vo, with Engravings, 5s.

Short Manual of Orthopædy. By HEATHER BIGG, F.R.C.S. Ed. Part I. Deformities and Deficiencies of the Head and Neck. 8vo. 2s. 6d.

Face and Foot Deformities. By FREDERICK CHURCHILL, C.M. 8vo, with Plates and Illustrations, 10s. 6d.

The Human Foot:
Its Form and Structure, Functions and Clothing. By THOMAS S. ELLIS, Consulting Surgeon to the Gloucester Infirmary. With 7 Plates and Engravings (50 Figures). 8vo, 7s. 6d.

Royal London Ophthalmic Hospital Reports. By the Medical and Surgical Staff. Vol. XIII., Part 3. 8vo, 5s.

Ophthalmological Society
of the United Kingdom. Transactions, Vol. XII. 8vo, 12s. 6d.

The Diseases of the Eye
(Student's Guide Series). By EDWARD NETTLESHIP, F.R.C.S., Ophthalmic Surgeon to St. Thomas's Hospital. Fifth Edition. Fcap. 8vo, with 164 Engravings and a Coloured Plate illustrating Colour-Blindness, 7s. 6d.

Diseases and Refraction of the Eye. By N. C. MACNAMARA, F.R.C.S., Surgeon to Westminster Hospital, and GUSTAVUS HARTRIDGE, F.R.C.S., Surgeon to the Royal Westminster Ophthalmic Hospital. Fifth Edition. Crown 8vo, with Plate, 156 Engravings, also Test-types, 10s. 6d.

On Diseases and Injuries of the Eye: A Course of Systematic and Clinical Lectures to Students and Medical Practitioners. By J. R. WOLFE, M.D., F.R.C.S.E., Lecturer on Ophthalmic Medicine and Surgery in Anderson's College, Glasgow. With 10 Coloured Plates and 157 Wood Engravings. 8vo, £1 1s.

Normal and Pathological Histology of the Human Eye and Eyelids. By C. FRED. POLLOCK, M.D., F.R.C.S. and F.R.S.E., Surgeon for Diseases of the Eye to Anderson's College Dispensary, Glasgow. Crown 8vo, with 100 Plates (230 drawings), 15s.

Refraction of the Eye:
A Manual for Students. By GUSTAVUS HARTRIDGE, F.R.C.S., Surgeon to the Royal Westminster Ophthalmic Hospital. Sixth Edition. Crown 8vo, with 98 Illustrations, also Test-types, &c., 6s.

By the same Author.

The Ophthalmoscope. A Manual for Students. Crown 8vo, with 63 Illustrations. 4s.

Atlas of Ophthalmoscopy.
Composed of 12 Chromo-lithographic Plates (59 Figures drawn from nature) and Explanatory Text. By RICHARD LIEBREICH, M.R.C.S. Translated by H. ROSBOROUGH SWANZY, M.B. Third edition, 4to, 40s.

Glaucoma:
Its Pathology and Treatment. By PRIESTLEY SMITH, Ophthalmic Surgeon to, and Clinical Lecturer on Ophthalmology at, the Queen's Hospital, Birmingham. 8vo, with 64 Engravings and 12 Zinco-photographs, 7s. 6d.

Eyestrain
(commonly called Asthenopia). By ERNEST CLARKE, M.D., B.S. Lond., Surgeon to the Central London Ophthalmic Hospital, Surgeon and Ophthalmic Surgeon to the Miller Hospital. 8vo, with 22 Illustrations, 5s.

Diseases of the Eye:
A Handbook of Ophthalmic Practice for Students and Practitioners. By G. E. DE SCHWEINITZ, M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic. With 216 Illustrations, and 2 Chromo-Lithographic Plates. 8vo, 18s.

Diseases and Injuries of the Ear. By Sir WILLIAM B. DALBY, F.R.C.S., M.B., Consulting Aural Surgeon to St. George's Hospital. Fourth Edition. Crown 8vo, with 8 Coloured Plates and 38 Wood Engravings. 10s. 6d.

By the same Author.

Short Contributions to Aural Surgery, between 1875 and 1889. Second Edition. 8vo, with Engravings, 3s. 6d.

Sore Throat:
Its Nature, Varieties, and Treatment. By PROSSER JAMES, M.D., Physician to the Hospital for Diseases of the Throat. Fifth Edition. Post 8vo, with Coloured Plates and Engravings, 6s. 6d.

Endemic Goitre or Thyreocele:
Its Etiology, Clinical Characters, Pathology, Distribution, Relations to Cretinism, Myxœdema, &c., and Treatment. By WILLIAM ROBINSON, M.D. 8vo, 5s.

Hints on Ophthalmic Out-Patient Practice. By CHARLES HIGGENS, Ophthalmic Surgeon to Guy's Hospital. Third Edition. Fcap. 8vo, 3s.

A System of Dental Surgery.
By Sir JOHN TOMES, F.R.S., and C. S. TOMES, M.A., F.R.S. Third Edition. Crown 8vo, with 292 Engravings, 15s.

Dental Anatomy, Human and Comparative: A Manual. By CHARLES S. TOMES, M.A., F.R.S. Third Edition. Crown 8vo, with 212 Engravings, 12s. 6d.

A Manual of Nitrous Oxide Anæsthesia, for the use of Students and General Practitioners. By J. FREDERICK W. SILK, M.D. Lond., M.R.C.S., Anæsthetist to the Royal Free Hospital, Dental School of Guy's Hospital, and National Epileptic Hospital. 8vo, with 26 Engravings, 5s.

A Practical Treatise on Mechanical Dentistry. By JOSEPH RICHARDSON, M.D., D.D.S., late Emeritus Professor of Prosthetic Dentistry in the Indiana Medical College. Fifth Edition. Roy. 8vo, with 458 Engravings, 21s.

Notes on Dental Practice.
By HENRY C. QUINBY, L.D.S.I., President-Elect of the British Dental Association. Second Edition. 8vo, with 92 Illustrations, 8s.

Elements of Dental Materia Medica and Therapeutics, with Pharmacopœia. By JAMES STOCKEN, L.D.S.R.C.S., Pereira Prizeman for Materia Medica, and THOMAS GADDES, L.D.S. Eng. and Edin. Third Edition. Fcap. 8vo, 7s. 6d.

Papers on Dermatology.
By E. D. MAPOTHER, M.D., Ex-Pres. R.C.S.I. 8vo, 3s. 6d.

Atlas of Skin Diseases.
By TILBURY FOX, M.D., F.R.C.P. With 72 Coloured Plates. Royal 4to, half morocco, £6 6s.

Diseases of the Skin:
A Practical Treatise for the Use of Students and Practitioners. By J. N. HYDE, A.M., M.D., Professor of Skin and Venereal Diseases, Rush Medical College, Chicago. Second Edition. 8vo, with 2 Coloured Plates and 96 Engravings, 20s.

Leprosy in British Guiana.
By JOHN D. HILLIS, F.R.C.S., M.R.I.A., Medical Superintendent of the Leper Asylum, British Guiana. Imp. 8vo, with 22 Lithographic Coloured Plates and Wood Engravings, £1 11s. 6d.

Diseases of the Skin
(Introduction to the Study of). By P. H. PYE-SMITH, M.D., F.R.S., F.R.C.P., Physician to, and Leeturer on Medieine in, Guy's Hospital. Crown 8vo, with 26 Engravings. 7s. 6d.

Sarcoma and Carcinoma:
Their Pathology, Diagnosis, and Treatment. By HENRY T. BUTLIN, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital. 8vo, with 4 Plates, 8s.

By the same Author.

Malignant Disease of the Larynx (Sarcoma and Carcinoma). 8vo, with 5 Engravings, 5s.

Also.

Operative Surgery of Malignant Disease. 8vo, 14s.

On Cancer:
Its Allies, and other Tumours; their Medieal and Surgieal Treatment. By F. A. PURCELL, M.D., M.C., Surgeon to the Caneer Hospital, Brompton. 8vo, with 21 Engravings, 10s. 6d.

Cancers and the Cancer Process: a Treatise, Praetical and Theoretie. By HERBERT L. SNOW, M.D., Surgeon to the Caneer Hospital, Brompton. 8vo, with 15 Lithographic Plates. 15s.

By the same Author.

The Re-appearance (Recurrence) of Cancer after apparent Extirpation. 8vo, 5s. 6d.

Also,

The Palliative Treatment of Incurable Cancer. Crown 8vo, 2s. 6d.

Cancerous Affections of the Skin.
(Epithelioma and Rodent Uleer.) By GEORGE THIN, M.D. Post 8vo, with 8 Engravings, 5s.

By the same Author.

Pathology and Treatment of Ringworm. 8vo, with 21 Engravings, 5s.

Diagnosis and Treatment of Syphilis. By TOM ROBINSON, M.D., Physician to St. John's Hospital for Diseases of the Skin. Crown 8vo, 3s. 6d.

By the same Author.

Eczema: its Etiology, Pathology, and Treatment. Crown 8vo, 3s. 6d.

Also.

Illustrations of Diseases of the Skin and Syphilis, with Remarks. Fase. I. with 3 Plates. Imp. 4to, 5s.

Diseases of the Urinary Organs.
Clinieal Lectures. By Sir HENRY THOMPSON, F.R.C.S., Emeritus Professor of Clinical Surgery and Consulting Surgeon to University College Hospital. Eighth Edition. 8vo, with 121 Engravings, 10s. 6d.

By the same Author.

Diseases of the Prostate:
Their Pathology and Treatment. Sixth Edition. 8vo, with 39 Engravings, 6s.

Also.

Surgery of the Urinary Organs.
Some Important Points eonneeted therewith. Lectures delivered in the R.C.S. 8vo, with 44 Engravings. Student's Edition, 2s. 6d.

Also.

Practical Lithotomy and Lithotrity; or, An Inquiry into the Best Modes of Removing Stone from the Bladder. Third Edition. 8vo, with 87 Engravings, 10s.

Also.

The Preventive Treatment of Calculous Disease, and the Use of Solvent Remedies. Third Edition. Crown 8vo, 2s. 6d.

Also.

Tumours of the Bladder:
Their Nature, Symptoms, and Surgieal Treatment. 8vo, with numerous Illustrations, 5s.

Also.

Stricture of the Urethra, and Urinary Fistulæ: their Pathology and Treatment. Fourth Edition. 8vo, with 74 Engravings, 6s.

Also.

The Suprapubic Operation of Opening the Bladder for the Stone and for Tumours. 8vo, with 14 Engravings, 3s. 6d.

Also.

Introduction to the Catalogue;
being Notes of a Thousand Cases of Calculi of the Bladder removed by the Author and now in the Hunterian Museum of the Royal College of Surgeons. 8vo, 2s. 6d.

Electric Illumination of the Bladder and Urethra, as a Means of Diagnosis of Obseure Vesico-Urethral Diseases. By E. HURRY FENWICK, F.R.C.S., Surgeon to London Hospital and St. Peter's Hospital for Stone. Second Edition. 8vo, with 54 Engravings, 6s. 6d.

By the same Author.

The Cardinal Symptoms of Urinary Diseases: their Diagnostic Significance and Treatment. 8vo, with 36 Illustrations. 8s. 6d.

Surgical Disorders of the Urinary Organs (Lectures). By REGINALD HARRISON, F.R.C.S., Surgeon to St. Peter's Hospital. Fourth Edition. 8vo, largely Illustrated, 16s.

Atlas of Electric Cystoscopy. By Dr. EMIL BURCKHARDT, late of the Surgical Clinique of the University of Bâle, and E. HURRY FENWICK, F.R.C.S., Surgeon to the London Hospital and St. Peter's Hospital for Stone. Royal 8vo, with 34 Coloured Plates, embracing 83 Figures. 21s.

Clinical Chemistry of Urine (Outlines of the). By C. A. MACMUNN, M.A., M.D. 8vo, with 64 Engravings and Plate of Spectra, 9s.

Urinary and Renal Derangements and Calculous Disorders. By LIONEL S. BEALE, F.R.C.P., F.R.S., Physician to King's College Hospital. 8vo, 5s.

Male Organs of Generation (Diseases of). By W. H. A. JACOBSON, M.Ch. Oxon., F.R.C.S., Assistant Surgeon to Guy's Hospital. 8vo, with 88 Engravings. 22s.

The Surgical Diseases of the Genito-Urinary Organs, including Syphilis. By E. L. KEYES, M.D., Professor in Bellevue Hospital Medical College, New York (a revision of VAN BUREN and KEYES' Text-book). Roy. 8vo, with 114 Engravings, 21s.

Diseases of the Rectum and Anus. By ALFRED COOPER, F.R.C.S., Senior Surgeon to the St. Mark's Hospital for Fistula; and F. SWINFORD EDWARDS, F.R.C.S., Senior Assistant Surgeon to St. Mark's Hospital. Second Edition, with Illustrations. 8vo, 12s.

Diseases of the Rectum and Anus. By HARRISON CRIPPS, F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital, &c. Second Edition. 8vo, with 13 Lithographic Plates and numerous Wood Engravings, 12s. 6d.

By the same Author.

Cancer of the Rectum. Especially considered with regard to its Surgical Treatment. Jacksonian Prize Essay. 8vo, with 13 Plates and several Wood Engravings, 6s.

The Diagnosis and Treatment of Diseases of the Rectum. By WILLIAM ALLINGHAM, F.R.C.S., Surgeon to St. Mark's Hospital for Fistula. Fifth Edition. By HERBERT WM. ALLINGHAM, F.R.C.S., Surgeon to the Great Northern Central Hospital, Demonstrator of Anatomy at St. George's Hospital. 8vo, with 53 Engravings. 10s. 6d.

The Surgery of the Rectum. By HENRY SMITH, Emeritus Professor of Surgery in King's College, Consulting Surgeon to the Hospital. Fifth Edition. 8vo, 6s.

A Medical Vocabulary: An Explanation of all Terms and Phrases used in the various Departments of Medical Science and Practice, their Derivation, Meaning, Application, and Pronunciation. By R. G. MAYNE, M.D., LL.D. Sixth Edition by W. W. WAGSTAFFE, B.A., F.R.C.S. Crown 8vo, 10s. 6d.

A Short Dictionary of Medical Terms. Being an Abridgment of Mayne's Vocabulary. 64mo, 2s. 6d.

Terminologia Medica Polyglotta: a Concise International Dictionary of Medical Terms (French, Latin, English, German, Italian, Spanish, and Russian). By THEODORE MAXWELL, M.D., B.Sc., F.R.C.S. Edin. Royal 8vo, 16s.

A German-English Dictionary of Medical Terms. By FREDERICK TREVES, F.R.C.S., Surgeon to the London Hospital; and HUGO LANG, B.A. Crown 8vo, half-Persian calf, 12s.

The Medical Adviser in Life Assurance. By Sir E. H. SIEVEKING, M.D., F.R.C.P. Second Edition. Crown 8vo, 6s.

Chemistry, Inorganic and Organic. With Experiments. By CHARLES L. BLOXAM. Seventh Edition, by JOHN MILLAR THOMSON, Professor of Chemistry in King's College, London, and ARTHUR G. BLOXAM, Demonstrator of Chemistry, in the Royal Agricultural College, Cirencester. 8vo, with 282 Illustrations, 18s.

By the same Author.

Laboratory Teaching; Or, Progressive Exercises in Practical Chemistry. Fifth Edition. Crown 8vo, with 89 Engravings, 5s. 6d.

Practical Chemistry And Qualitative Analysis. By FRANK CLOWES, D.Sc. Lond., Professor of Chemistry in the University College, Nottingham. Fifth Edition. Post 8vo, with 57 Engravings and Frontispiece, 7s. 6d.

Quantitative Analysis. By FRANK CLOWES, D.Sc. Lond., Professor of Chemistry in the University College, Nottingham, and J. BERNARD COLEMAN, Assoc. R. C. Sci. Dublin; Senior Demonstrator of Chemistry in the University College, Nottingham. Post 8vo, with 83 Engravings, 7s. 6d.

Watts' Manual of Chemistry,
Theoretical and Practical. By WILLIAM A. TILDEN, D.Sc., F.R.S., Professor of Chemistry in the Mason College, Birmingham.

PHYSICAL AND INORGANIC CHEMISTRY. Second Edition. Crown 8vo, with Coloured Plate of Spectra, and 122 Wood Engravings, 8s. 6d.

CHEMISTRY OF CARBON COMPOUNDS; or, ORGANIC CHEMISTRY. Second Edition. Crown 8vo, with Engravings, 10s.

Qualitative Analysis.
By R. FRESENIUS. Translated by CHARLES E. GROVES, F.R.S. Tenth Edition. 8vo, with Coloured Plate of Spectra and 46 Engravings, 15s.

By the same Author.

Quantitative Analysis.
Seventh Edition.
Vol. I., Translated by A. VACHER. 8vo, with 106 Engravings, 15s.
Vol. II., Parts 1 to 3, Translated by C. E. GROVES, F.R.S. 8vo, with Engravings, 2s. 6d. each.

Practical Chemistry,
Including Analysis. By John E. BOWMAN and CHARLES L. BLOXAM. Fcap. 8vo. Eighth Edition, with 90 Engravings, 5s. 6d.

Inorganic Chemistry.
By EDWARD FRANKLAND, Ph.D., D.C.L., LL.D., F.R.S., Professor of Chemistry in the Normal School of Science, and FRANCIS R. JAPP, M.A., Ph.D. F.I.C., F.R.S., Professor of Chemistry in the University of Aberdeen. 8vo, with numerous Illustrations on Stone and Wood, 24s.

Inorganic Chemistry
(A System of). By WILLIAM RAMSEY, Ph.D., F.R.S., Professor of Chemistry in University College, London. 8vo, with Engravings, 15s.

By the same Author.

Elementary Systematic Chemistry for the Use of Schools and Colleges. With Engravings. Crown 8vo, 4s. 6d.; Interleaved, 5s. 6d.

Organic Chemistry:
(Outlines of). By H. FORSTER MORLEY, M.A., D.Sc., Joint Editor of Watts' "Dictionary of Chemistry." Crown 8vo, 7s. 6d.

Valentin's Qualitative Chemical Analysis. Eighth Edition. By W. R. HODGKINSON, Ph.D., F.R.S.E., Professor of Chemistry and Physics in the Royal Military Academy and Artillery College, Woolwich. 8vo, with Engravings and Map of Spectra, 8s. 6d.

Analytical Chemistry.
Notes for Students in Medicine. By ALBERT J. BERNAYS, Ph.D., F.C.S., F.I.C., late Professor of Chemistry, &c., at St. Thomas's Hospital Medical School. Third Edition. Crown 8vo, 4s. 6d.

Volumetric Analysis:
(A Systematic Handbook of); or the Quantitative Estimation of Chemical Substances by Measure, applied to Liquids, Solids, and Gases. By FRANCIS SUTTON, F.C.S., F.I.C., Public Analyst for the County of Norfolk. Sixth Edition. 8vo, with 102 Engravings, 17s. 6d.

Commercial Organic Analysis:
A Treatise on the Properties, Modes of Assaying, Proximate Analytical Examination, &c., of the various Organic Chemicals and Products employed in the Arts, Manufactures, Medicine, &c. By ALFRED H. ALLEN, F.I.C., F.C.S., Public Analyst for the West Riding of Yorkshire, the Northern Division of Derbyshire, &c.

Vol. I.—Alcohols, Neutral Alcoholic Derivatives, Sugars, Starch and its Isomers, Vegetable Acids, &c. With Illustrations. Third Edition. 8vo. [*Preparing.*

Vol. II.—Fixed Oils and Fats, Hydrocarbons, Phenols, &c. With Illustrations. Third Edition. 8vo. [*Preparing.*

Vol. III.—Part I. Aromatic Acids, Tannins, Dyes, and Colouring Matters. Second Edition, 8vo, 14s.

Part II. Amines and Ammonium Bases, Hydrazines, Bases from Tar, Vegetable Alkaloids. Second Edition. 8vo, 18s.

Cooley's Cyclopædia
of Practical Receipts, and Collateral Information in the Arts, Manufactures, Professions, and Trades: Including Medicine, Pharmacy, Hygiene and Domestic Economy. Seventh Edition, by W. NORTH, M.A. Camb., F.C.S. 2 Vols., Roy. 8vo, with 371 Engravings, 42s.

Chemical Technology:
A Manual. By RUDOLF VON WAGNER. Translated and Edited by WILLIAM CROOKES, F.R.S., from the Thirteenth Enlarged German Edition as remodelled by Dr. FERDINAND FISCHER. 8vo, with 596 Engravings, 32s.

Fuel and its Applications.
By E. J. MILLS, D.Sc., F.R.S., and F. J. ROWAN, C.E. Being Vol. I. of Chemical Technology, or Chemistry in its Application to Arts and Manufactures. Edited by CHARLES E. GROVES, F.R.S., and WILLIAM THORP, B.Sc. Royal 8vo, with 606 Engravings, 30s.

Potable Waters:
Their Organic Analysis. By J. A. BLAIR, M.B., C.M., D.Sc., L.R.C.P. Second Edition. Crown 8vo, 3s. 6d.

Technological Handbooks.
EDITED BY JOHN GARDNER, F.I.C., F.C.S., and JAMES CAMERON, F.I.C.

BREWING, DISTILLING, AND WINE MANUFACTURE. Crown 8vo, with Engravings, 6s. 6d.

BLEACHING, DYEING, AND CALICO PRINTING. With Formulæ. Crown 8vo, with Engravings, 5s.

OILS, RESINS, AND VARNISHES. Crown 8vo, with Engravings. 7s. 6d.

SOAPS AND CANDLES. Crown 8vo, with 54 Engravings, 7s.

The Microscope and its Revelations. By the late WILLIAM B. CARPENTER, C.B., M.D., LL.D., F.R.S. Seventh Edition, by the Rev. W. H. DALLINGER, LL.D., F.R.S. With 21 Plates and 800 Wood Engravings. 8vo, 26s. Half Calf, 30s.

The Quarterly Journal of Microscopical Science. Edited by E. RAY LANKESTER, M.A., LL.D., F.R.S.; with the co-operation of ADAM SEDGWICK, M.A., F.R.S., A. MILNES MARSHALL, M.A., D.Sc., M.D., F.R.S., and W. F. R. WELDON, M.A., F.R.S. Each Number, 10s.

Methods and Formulæ
Used in the Preparation of Animal and Vegetable Tissues for Microscopical Examination, including the Staining of Bacteria. By PETER WYATT SQUIRE, F.L.S. Crown 8vo, 3s. 6d.

The Microtomist's Vade-Mecum:
A Handbook of the Methods of Microscopic Anatomy. By ARTHUR BOLLES LEE, Assistant in the Russian Laboratory of Zoology at Villefranche-sur-mer (Nice). Third Edition. 8vo, 14s.

Photo-Micrography
(Guide to the Science of). By EDWARD C. BOUSFIELD, L.R.C.P. Lond. 8vo, with 34 Engravings and Frontispiece, 6s.

The Principles and Practice of Veterinary Medicine. By WILLIAM WILLIAMS, F.R.C.V.S., F.R.S.E., Principal, and Professor of Veterinary Medicine and Surgery at the New Veterinary College, Edinburgh. Seventh Edition. 8vo, with several Coloured Plates and Woodcuts, 30s.

By the same Author.

The Principles and Practice of Veterinary Surgery. Eighth Edition. 8vo, with 9 Plates and 147 Woodcuts, 30s.

A Pharmacopœia, including the Outlines of Materia Medica and Therapeutics, for the Use of Practitioners and Students of Veterinary Medicine. By RICHARD V. TUSON, F.I.C., late Professor of Chemistry, Materia Medica, and Toxicology at the Royal Veterinary College. Fourth Edition. Post 8vo, 7s. 6d.

The Veterinarian's Pocket Remembrancer: being Concise Directions for the Treatment of Urgent or Rare Cases, embracing Semeiology, Diagnosis, Prognosis, Surgery, Therapeutics, Toxicology, Detection of Poisons by their Appropriate Tests, Hygiene, &c. By GEORGE ARMATAGE, M.R.C.V.S. Second Edition. Post 8vo, 3s.

Chauveau's Comparative Anatomy of the Domesticated Animals. Revised and Enlarged, with the Co-operation of S. ARLOING, Director of the Lyons Veterinary School, and Edited by GEORGE FLEMING, C.B., LL.D., F.R.C.V.S., late Principal Veterinary Surgeon of the British Army. Second English Edition. 8vo, with 585 Engravings, 31s. 6d.

INDEX TO J. & A. CHURCHILL'S LIST.

[*Continued on the next page.*

N.B.—J. & A. Churchill's larger Catalogue of about 600 *works on Anatomy, Physiology, Hygiene, Midwifery, Materia Medica, Medicine, Surgery, Chemistry, Botany, &c., &c., with a complete Index to their Subjects, for easy reference, will be forwarded post free on application.*

AMERICA.—*J. & A. Churchill being in constant communication with various publishing houses in America are able to conduct negotiations favourable to English Authors.*